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DIFFERENTIAL FERTILITY IN RELATION TO BODY DIMENSIONS

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INTRODUCTION

ALTHOUGH differential fertility has been frequently studied in relation to socio-economic and educational factors, few studies have been directed at the relationship between physical characteristics and fertility. This is the more surprising in view of the fact that several studies (for example, Newman *et al.*, 1937, and Clark, 1956) have shown that variation in body measurements is to a large extent under genetic control and that accordingly a relationship between these measurements and fertility could have a bearing on the rate and direction of human evolution.

In 1950 the Institute of Human Biology of the University of Michigan initiated the Assortative Mating Study. The major objective of this study has been to ascertain the genetic consequences of assortative mating in a human population. The study is consequently concerned with the

relations, if any, between fertility and the various physical, psychological, and social attributes of man. The present paper consists of a preliminary analysis of the measurements and information obtained by the study which pertain to the relations between fertility and certain physical characters.

ACKNOWLEDGMENTS

The Assortative Mating Study was supported from 1950 to 1955 by a generous grant to the University of Michigan from an anonymous donor. The field and laboratory work of the Study were conducted by the Institute of Human Biology under the direction of Lee R. Dice. The statistical computations for this paper were completed while the senior author was employed during the summer of 1958 by Cranbrook Institute of Science under a grant (M-1650) from the National Institute of Mental Health.

THE SAMPLE

The individuals whose measurements and fertility are analyzed in this paper were selected at random from the adult population of Ann Arbor, Michigan, in two stages. Males were considered to be adult if they were at least 18 years old; females if they were at least 17 years old. The primary sampling units were defined in terms of number of dwelling units, such that each primary unit contained at least 10 and at most 20 dwelling units. The city of Ann Arbor was divided into 699 such primary sampling units, and of these 108 were selected at random in the first stage of the sampling. A field census was then made of all the individuals resident in each primary unit selected for study. The individuals were classified into married pairs, single females, and single males. A single individual was defined as one not married at the time of the census, regardless of whether or not he or she had previously been married. From each of these three categories selections were made at random, as follows:

1. Married pairs, selection rate $\frac{1}{4}$
2. Single females, selection rate $\frac{1}{4}$
3. Single males, selection rate $\frac{1}{2}$.

The selection rate for single males was made larger than that for single females because there were fewer single males than single females in the population.

The selections were made during the years 1951-1954, inclusive. Every

effort was made to make the sample as random as possible. However, inasmuch as about a quarter of the individuals selected for study refused to participate, the sample cannot be regarded as wholly random. Only persons of European descent were used in this aspect of the study. Females less than 20 and males less than 25 years of age, although present in the sample, were not included in this study because they exhibited almost no variation in fertility.

The complete sample used in this study consisted of 97 single females and 227 married females, 43 single males and 187 married males.

FERTILITY

The term fertility, as employed in this paper, pertains to the total number of children which an individual acknowledges to have produced. It has no reference to innate reproductive capacity or to the multitudinous factors underlying variation in reproductive performance. Our concern here is entirely with actual number of children produced, as it is this factor which is most relevant to the future genetic composition of the population. Consequently, in investigating the relationship between fertility and body measurements, single and married individuals are combined in the same sample.

Another reason for ignoring marital status in the present study is the considerable difficulty in accounting for it. It is obviously not sufficient merely to classify people as married or not married, for individuals differ in the length of time they have been married. Moreover, the significance, so far as fertility is concerned, of a year of married life depends on the age of the individuals at the time. A year of marriage commencing at age 20, for example, is more likely to result in the birth of a child than is a year of marriage commencing at age 50. Furthermore, the importance of a year of marriage even for individuals of the same age varies with their economic condition at the time. These considerations, combined with the fact that many individuals have been married several times, make it exceedingly difficult to take this factor into consideration in studying fertility. And besides, as pointed out in the previous paragraph, it is not necessary to consider it so far as the genetic and evolutionary implications of the data are concerned.

The 324 females were classified into 11 age groups and the 230 males into 9 age groups. The fertility of each individual was then classified as either above average or below average, depending upon whether the number of children acknowledged by the individual was greater than or

TABLE 1

Age and fertility categories for males, showing the number of single and married individuals in each of the 18 categories, the mean fertility per individual and the range of fertility per individual for each category

	BELOW AVERAGE FERTILITY				ABOVE AVERAGE FERTILITY			
	NUMBER OF INDIVIDUALS		MEAN FERTILITY	RANGE OF FERTILITY	NUMBER OF INDIVIDUALS		MEAN FERTILITY	RANGE OF FERTILITY
AGE	SINGLE	MARRIED			SINGLE	MARRIED		
25–29	8	20	0.0		0	25	1.4	1–2
30–34	3	17	0.4	0–1	0	17	2.5	2–4
35–39	2	7	0.3	0–1	0	14	2.6	2–4
40–44	1	6	0.4	0–1	0	9	2.4	2–4
45–49	0	13	1.1	0–2	0	8	3.2	3–4
50–54	6	4	0.1	0–1	1	12	2.9	2–4
55–59	3	4	0.6	0–1	1	12	2.6	2–4
60–69	7	3	0.4	0–2	3	8	4.1	3–6
70–	5	3	0.4	0–1	3	5	3.9	2–6

TABLE 2

Age and fertility categories for females, showing the number of single and married individuals in each of the 22 categories, the mean fertility per individual and the range of fertility per individual for each category.

	BELOW AVERAGE FERTILITY				ABOVE AVERAGE FERTILITY			
	NUMBER OF INDIVIDUALS		MEAN FERTILITY	RANGE OF FERTILITY	NUMBER OF INDIVIDUALS		MEAN FERTILITY	RANGE OF FERTILITY
AGE	SINGLE	MARRIED			SINGLE	MARRIED		
20–24	14	19	0.0		1	18	1.4	1–2
25–29	12	21	0.0		1	29	1.6	1–3
30–34	4	7	0.5	0–1	0	20	2.7	2–4
35–39	7	5	0.3	0–1	1	12	3.2	2–6
40–44	4	6	0.0		1	13	2.1	1–4
45–49	8	13	0.5	0–1	1	22	2.7	2–5
50–54	2	6	0.6	0–1	1	15	2.9	2–7
55–59	2	4	0.7	0–2	2	5	4.0	3–8
60–64	4	3	1.0	0–2	2	3	4.6	3–6
65–69	6	2	1.5	0–2	8	1	5.1	3–12
70–	10	1	0.9	0–2	6	2	4.0	3–7

less than the median for its sex and age group. In total, therefore, the males were classified into 18 and the females into 22 categories. These categories, together with the mean and the range of the number of children per individual and the number of single and married individuals in each, are shown for males and females in tables 1 and 2, respectively. It may be seen from these tables that the largest number of children reported by any male was 6 and that the most fertile female produced 12 children. The lowest number of children produced was zero for both sexes. Taken as a whole, the sample can scarcely be regarded as a highly fertile one.

MEASUREMENTS

All anthropometric measurements were made in the anthropometry room of the University of Michigan's Institute of Human Biology. Measurements were taken on the subject with shoes and clothing external to shirts and blouses removed. The sleeves of shirts or blouses were rolled up in order to make the various arm measurements, and pant legs or skirts were elevated in order to make measurements on the lower leg.

The measurements were made with an anthropometer, sliding caliper, spreading caliper, Hrdlička caliper, steel metric tape, and with platform scales calibrated in quarter pounds. Wall-mounted scales of standard metric sticks were used to measure stature and span. A low bench with a vertical scale was used to measure sitting height.

The methods of taking the measurements are those of Martin (1928). The measurements used in this aspect of the study, together with the instruments employed and the numbers by which Martin designated the measurements, are given below.

MEASUREMENTS OF BODY AND EXTREMITIES

Net weight. Platform scales. Gross weight (Martin 71) minus estimated weight of clothes.

Stature. Wall scale (Martin 1).

Cervical height. Anthropometer. Height from the floor of the apex of the tip of the spinous process of the seventh cervical vertebra.

Span. Wall scale (Martin 17).

Sitting height. Sitting height scale (Martin 23).

Biacromial breadth. Anthropometer used as a sliding caliper (Martin 35).

Forearm length. Anthropometer used as a sliding caliper (Martin 48).

Hand length. Sliding caliper (Martin 49).

Middle finger length. Sliding caliper (Martin 51).
Hand breadth. Sliding caliper (Martin 52).
Foot length. Anthropometer used as a sliding caliper (Martin 58).
Minimum neck circumference. Steel tape, just below thyroid cartilage (Martin 63).
Maximum midarm circumference. Steel tape (Martin 65).
Maximum forearm circumference. Steel tape (Martin 66).
Minimum wrist circumference. Steel tape (Martin 67).
Maximum calf circumference. Steel tape (Martin 69).
Minimum ankle circumference. Steel tape (Martin 70).
Bicondylar breadth of elbow. Hrdlička caliper.
Bicondylar breadth of knee. Hrdlička caliper.

MEASUREMENTS OF HEAD AND FACE

Head length. Spreading caliper (Martin 1).
Head breadth. Spreading caliper (Martin 3).
Head height. Anthropometer used as a sliding caliper (Martin 15).
Minimum frontal breadth. Spreading caliper (Martin 4).
Bizygomatic breadth. Spreading caliper (Martin 6).
Bigonial breadth. Spreading caliper Martin 8).
Bipalpebral breadth. Sliding caliper (Martin 10).
Interpupillary distance. Sliding caliper (Martin 12).
Nose height. Sliding caliper (Martin 21).
Nose breadth. Sliding caliper (Martin 13).
Total facial height. Sliding caliper (Martin 18).
Ear height. Sliding caliper (Martin 29).
Ear lobe height. Sliding caliper (Martin 33).

In addition to the direct measurements listed above, the following ratios were also investigated:

$$\frac{\text{stature}}{\text{span}}, \quad \frac{\text{sitting height}}{\text{stature}}, \quad \frac{\text{shoulder breadth}}{\text{stature}}, \quad \frac{\text{hand breadth}}{\text{hand length}},$$

$$\frac{\text{head breadth}}{\text{head length}}, \quad \frac{\text{bizygomatic breadth}}{\text{head breadth}}, \quad \frac{\text{bigonal breadth}}{\text{minimum frontal breadth}},$$

$$\frac{\text{nose height}}{\text{total facial height}}, \text{ and } \frac{\text{nose breadth}}{\text{nose height}}.$$

Most of the measurements were performed by the authors. Measurements were also taken by Neil Tappen, Van Harris, and Jack Kelso.

ANALYSES

The relationships between the anthropometric traits and fertility were analyzed separately for each trait and each sex by a series of $r \times 2$ analyses of variance, there being r age groups (with $r = 9$ for males; $r = 11$ for females) and two fertility classes (namely, above average and below average fertility) for each analysis. Since 41 characters were investigated, this means that 82 separate analyses of variance were performed. The method of analysis is that described by Snedecor (1956, pp. 382-384) for disproportionate subclass numbers.

Although many of the anthropometric traits are conspicuously correlated with age, the investigation of these relationships is not within the scope of this paper. Individuals were classified into age groups only in order to prevent the relationships with age from being confounded with those between the anthropometric traits and fertility, the latter relationships being the only ones under consideration in this paper.

RESULTS

A summary of the results of the analyses is given in tables 3 and 4 for males and females, respectively. These tables include for each trait an estimate, D, of the difference in the mean value of the trait between the above average and the below average fertility groups (*i.e.*, above average minus below average fertility), the F-value for the relation between the trait and fertility, the F-value for the effect on the trait of the interaction between age and fertility, and the probabilities of obtaining F-values as large as those observed on the assumption that the anthropometric traits and fertility are independent of one another. In discussing the various traits a single asterisk will indicate significance at the 5% level; two asterisks will indicate significance at the 1% level.

Examination of table 3 reveals that in the males the F-values for fertility were significant for the following characters: Head length*, bizygomatic breadth*, minimum neck circumference**, weight**, biacromial breadth*, maximum forearm circumference*, maximum midarm circumference**, minimum wrist circumference**, maximum calf circumference**, bicondylar breadth of knee* and $\frac{\text{stature}^*}{\text{span}}$.

The first 10 of these characters involve direct measurements and were positively correlated with fertility. In other words, the character under question had a larger mean value in the more fertile than in the

TABLE 3. MALES

Results of Analyses of Variance for each of the 41 traits, showing an estimate, D, of the difference in the mean value of each trait between the above average and the below average fertility groups (i. e., above average fertility minus below average fertility), the F-value for the relationship between the trait and fertility, the F-value for the interaction between age and fertility, and the probabilities of obtaining F-values as large as those observed on the assumption that the anthropometric traits and fertility are independent of one another. The F for fertility contains 1 degree of freedom in the numerator and 212 in the denominator; that for interaction has 8 degrees of freedom in the numerator and 212 in the denominator.

CHARACTER	*D*	*F*-VALUE FOR FERTILITY	PROBABILITY OF LARGER *F*	*F*-VALUE FOR INTERACTION	PROBABILITY OF LARGER *F*
Weight	11.33 lb	10.30	$.005 > P > .001$	2.02	$.050 > P > .025$
Stature	—3.66 mm	0.18	$.75 > P > .50$	2.92	$.005 > P > .001$
Cervical height	0.89 mm	0.01	$.95 > P > .90$	0.78	$.75 > P > .50$
Span	13.92 mm	2.02	$.25 > P > .10$	1.01	$.50 > P > .30$
Sitting height	5.26 mm	1.48	$.25 > P > .10$	0.85	$.75 > P > .50$
Biacromial breadth	5.53 mm	3.97	$.050 > P > .025$	0.98	$.50 > P > .30$
Forearm length	—0.11 mm	0.004	$.975 > P > .950$	1.25	$.30 > P > .10$
Hand length	2.49 mm	1.50	$.25 > P > .10$	1.20	$.50 > P > .30$
Middle finger length	2.39 mm	2.95	$.10 > P > .05$	0.40	$.95 > P > .90$
Hand breadth	1.02 mm	3.11	$.10 > P > .05$	1.12	$.50 > P > .30$
Foot length	0.27 mm	0.03	$.90 > P > .75$	0.71	$.75 > P > .50$
Minimum neck circumference	9.90 mm	10.09	$.005 > P > .001$	1.52	$.25 > P > .10$
Maximum midarm circumference	13.44 mm	13.95	$.0005 > P$	2.77	$.010 > P > .005$
Maximum forearm circumference	5.67 mm	4.30	$.050 > P > .025$	2.63	$.010 > P > .005$
Minimum wrist circumference	4.37 mm	9.74	$.005 > P > .001$	1.91	$.10 > P > .05$
Maximum calf circumference	10.72 mm	8.73	$.005 > P > .001$	2.55	$.025 > P > .010$
Minimum ankle circumference	4.18 mm	2.77	$.10 > P > .05$	0.94	$.50 > P > .30$
Bicondylar breadth of elbow	1.56 mm	3.01	$.10 > P > .05$	1.59	$.30 > P > .10$
Bicondylar breadth of knee	1.48 mm	4.06	$.050 > P > .025$	1.34	$.25 > P > .10$
Head length	2.33 mm	6.11	$.025 > P > .010$	0.93	$.50 > P > .30$
Head breadth	0.83 mm	1.43	$.25 > P > .10$	1.21	$.30 > P > .10$
Head height	1.23 mm	1.52	$.25 > P > .10$	1.26	$.30 > P > .10$
Minimum frontal breadth	1.25 mm	3.44	$.10 > P > .05$	0.47	$.90 > P > .75$
Bizygomatic breadth	1.48 mm	4.33	$.050 > P > .025$	1.34	$.25 > P > .10$
Bigonial breadth	1.50 mm	3.47	$.10 > P > .05$	1.55	$.25 > P > .10$
Bipalpebral breadth	0.48 mm	0.66	$.50 > P > .25$	0.49	$.90 > P > .75$

TABLE 3. MALES (Continued)

CHARACTER	*D*	*F*-VALUE FOR FERTILITY	PROBABILITY OF LARGER *F*	*F*-VALUE FOR INTERACTION	PROBABILITY OF LARGER *F*
Interpupillary distance	0.23 mm	0.29	$.75>P>.50$	1.27	$.30>P>.10$
Nose height	0.36 mm	0.43	$.75>P>.50$	1.76	$.10>P>.05$
Nose breadth	—0.078 mm	0.04	$.90>P>.75$	0.92	$.75>P>.50$
Total facial height	—0.003 mm	0.001	$.975>P>.950$	1.07	$.50>P>.30$
Ear height	1.06 mm	3.67	$.10>P>.05$	1.62	$.25>P>.10$
Ear lobe height	0.13 mm	0.14	$.75>P>.50$	1.75	$.10>P>.05$
$\frac{\text{Stature}}{\text{Span}}$	—0.0054	4.00	$.050>P>.025$	1.04	$.50>P>.30$
$\frac{\text{Sitting height}}{\text{Stature}}$	0.0020	1.63	$.25>P>.10$	0.42	$.95>P>.90$
$\frac{\text{Shoulder breadth}}{\text{Stature}}$	0.0028	3.44	$.10>P>.05$	1.78	$.10>P>.05$
$\frac{\text{Hand breadth}}{\text{Hand length}}$	0.0071	2.10	$.25>P>.10$	0.82	$.75>P>.50$
$\frac{\text{Head breadth}}{\text{Head length}}$	—0.0052	1.14	$.50>P>.25$	1.41	$.25>P>.10$
$\frac{\text{Bizygomatic breadth}}{\text{Head breadth}}$	0.0049	1.53	$.25>P>.10$	0.92	$.75>P>.50$
$\frac{\text{Bigonial breadth}}{\text{Minimum frontal breadth}}$	0.0011	0.016	$.95>P>.90$	1.31	$.25>P>.10$
$\frac{\text{Nose height}}{\text{Total facial height}}$	0.0027	0.55	$.50>P>.25$	2.40	$.025>P>.010$
$\frac{\text{Nose breadth}}{\text{Nose height}}$	—0.0051	0.26	$.75>P>.50$	1.80	$.10>P>.05$

less fertile group. The last character, however, namely stature divided by span, was negatively correlated with fertility. Considering the entire group of 41 characters it will be seen that for males 28 of 32 measurements and 6 of the 9 ratios were positively related with fertility, although only for the characters previously listed did the *F*-values for fertility reach the 5% level of significance.

The *F*-values for the interaction between age and fertility were significant, among the males, for the following characters: Weight*, stature**, maximum forearm circumference**, maximum midarm circumference**, maximum calf circumference* and $\frac{\text{nose height}^*}{\text{total facial height}}$.

TABLE 4. FEMALES

Results of Analyses of Variance for each of the 41 traits, showing an estimate, D, of the difference in the mean value of each trait between the above average and the below average fertility groups (i. e., above average fertility minus below average fertility), the F-value for the relationship between the trait and fertility, the F-value for the interaction between age and fertility, and the probabilities of obtaining F-values as large as those observed on the assumption that the anthropometric traits and fertility are independent of one another. The F for fertility contains 1 degree of freedom in the numerator and 302 in the denominator; that for interaction contains 10 degrees of freedom in the numerator and 302 in the denominator.

CHARACTER	*D*	*F*-VALUE FOR FERTILITY	PROBABILITY OF LARGER *F*	*F*-VALUE FOR INTERACTION	PROBABILITY OF LARGER *F*
Weight	4.33 lb	2.51	$.25>P>.10$	0.54	$.90>P>.75$
Stature	—10.15 mm	2.14	$.25>P>.10$	0.20	$.999>P>.995$
Cervical height	—11.07 mm	2.93	$.10>P>.05$	0.41	$.95>P>.90$
Span	—3.38 mm	0.17	$.75>P>.50$	0.59	$.90>P>.75$
Sitting height	—7.54 mm	4.31	$.050>P>.025$	1.03	$.50>P>.30$
Biacromial breadth	0.70 mm	0.13	$.75>P>.50$	0.51	$.90>P>.75$
Forearm length	—0.28 mm	0.04	$.90>P>.75$	0.93	$.75>P>.50$
Hand length	0.24 mm	0.06	$.90>P>.75$	1.02	$.50>P>.30$
Middle finger length	0.53 mm	0.30	$.75>P>.50$	1.37	$.25>P>.10$
Hand breadth	0.98 mm	4.64	$.050>P>.025$	0.84	$.75>P>.50$
Foot length	0.14 mm	0.01	$.95>P>.90$	0.48	$.95>P>.90$
Minimum neck circumference	2.93 mm	1.68	$.25>P>.10$	0.99	$.50>P>.30$
Maximum midarm circumference	9.66 mm	6.46	$.025>P>.010$	0.82	$.75>P>.50$
Maximum forearm circumference	5.95 mm	7.81	$.010>P>.005$	1.17	$.50>P>.30$
Minimum wrist circumference	2.20 mm	4.06	$.050>P>.025$	1.06	$.50>P>.30$
Maximum calf circumference	4.34 mm	1.36	$.25>P>.10$	1.45	$.25>P>.10$
Minimum ankle circumference	3.85 mm	2.30	$.25>P>.10$	0.87	$.75>P>.50$
Bicondylar breadth, arm	1.11 mm	1.44	$.25>P>.10$	0.40	$.95>P>.90$
Bicondylar breadth, leg	0.79 mm	1.35	$.25>P>.10$	0.84	$.75>P>.50$
Head length	—.25 mm	0.12	$.75>P>.50$	0.35	$.975>P>.950$
Head breadth	—.18 mm	0.11	$.75>P>.50$	0.67	$.90>P>.75$
Head height	1.30 mm	2.45	$.25>P>.10$	1.18	$.50>P>.30$
Minimum frontal breadth	—0.55 mm	0.99	$.50>P>.25$	1.48	$.25>P>.10$
Bizygomatic breadth	0.32 mm	0.35	$.75>P>.50$	0.46	$.95>P>.90$
Bigonial breadth	—0.46 mm	0.63	$.50>P>.25$	0.54	$.90>P>.75$
Bipalpebral breadth	—0.21 mm	0.22	$.75>P>.50$	1.00	$.50>P>.30$

TABLE 4. FEMALES (Continued)

CHARACTER	*D*	*F*-VALUE FOR FERTILITY	PROBABILITY OF LARGER *F*	*F*-VALUE FOR INTERACTION	PROBABILITY OF LARGER *F*
Interpupillary distance	—0.53 mm	1.85	.25>*P*>.10	0.76	.75>*P*>.50
Nose height	—1.02 mm	7.18	.010>*P*>.005	0.76	.75>*P*>.50
Nose breadth	0.17 mm	0.39	.75>*P*>.50	1.19	.30>*P*>.10
Total facial height	—0.58 mm	0.71	.50>*P*>.25	1.25	.25>*P*>.10
Ear height	0.06 mm	0.02	.90>*P*>.75	0.92	.75>*P*>.50
Ear lobe height	—0.25 mm	0.55	.50>*P*>.25	2.18	.025>*P*>.010
$\frac{\text{Stature}}{\text{Span}}$	—0.0048	3.62	.10>*P*>.05	1.57	.25>*P*>.10
$\frac{\text{Sitting height}}{\text{Stature}}$	—.0010	0.38	.75>*P*>.50	0.86	.75>*P*>.50
$\frac{\text{Shoulder breadth}}{\text{Stature}}$	.0018	2.58	.25>*P*>.10	0.56	.90>*P*>.75
$\frac{\text{Hand breadth}}{\text{Hand length}}$	.0049	4.21	.050>*P*>.025	0.76	.75>*P*>.50
$\frac{\text{Head breadth}}{\text{Head length}}$	.0000	.00012	.975>*P*>.950	0.65	.90>*P*>.75
$\frac{\text{Bizygomatic breadth}}{\text{Head breadth}}$	.0031	1.03	.50>*P*>.25	1.27	.30>*P*>.10
$\frac{\text{Bigonial breadth}}{\text{Minimum frontal breadth}}$	.0006	0.01	.95>*P*>.90	1.29	.25>*P*>.10
$\frac{\text{Nose height}}{\text{Total facial height}}$	—.0086	6.96	.010>*P*>.005	0.58	.90>*P*>.75
$\frac{\text{Nose breadth}}{\text{Nose height}}$	.0156	4.26	.050>*P*>.025	1.35	.25>*P*>.10

For 4 of these characters, namely weight, maximum forearm circumference, maximum midarm circumference, and maximum calf circumference, the *F*-values for fertility were also significant. However, even though the main effect of fertility was not significant for stature or for $\frac{\text{nose height}}{\text{total facial height}}$, the existence for these traits of an interaction between age and fertility would be indicative of a relationship between the traits and fertility, although the nature of the relationship would vary from age group to age group.

From table 4 it may be seen that in the females the *F*-values for fertility are significant for the following characters: Nose height**,

sitting height*, maximum forearm circumference**, maximum midarm circumference*, minimum wrist circumference*, hand breadth*,

$$\frac{\text{hand breadth*}}{\text{hand length}}, \quad \frac{\text{nose breadth*}}{\text{nose height}}, \text{ and } \frac{\text{nose height**}}{\text{total facial height}}.$$

The first 6 of these characters involve direct measurements, and the last three are ratios of certain measurements. Of the direct measurements, the first two in the list, namely nose height and sitting height, were negatively correlated with fertility, and the last 4, namely maximum forearm circumference, maximum midarm circumference, minimum wrist circumference, and hand breadth, were positively correlated with fertility. In other words, the mean values for nose height and sitting height were larger in the less fertile than in the more fertile group, and the mean values for maximum forearm circumference, maximum midarm circumference, minimum wrist circumference, and hand breadth were larger for the more fertile than for the less fertile group. Of the three ratios for which the F-values for fertility were significant in the females, the last one, namely, $\frac{\text{nose height}}{\text{total facial height}}$, was negatively correlated with fertility, whereas $\frac{\text{hand breadth}}{\text{hand length}}$ and $\frac{\text{nose breadth}}{\text{nose height}}$ were positively correlated with fertility. Considering the entire group of 41 characters it may be seen that for females 18 of the 32 measurements and 6 of the 9 ratios were positively correlated with fertility, although only for the characters previously listed did the F-values for fertility reach the 5% level of significance.

The F-value for the interaction between age and fertility was significant at the 5% level, in the females, only for ear lobe height.

For the three characters maximum midarm circumferences, maximum forearm circumference, and minimum wrist circumference, the F-values for fertility were significant for both males and females; the character $\frac{\text{nose height}}{\text{total facial height}}$ gave a significant F for fertility in the females and a significant F for interaction between age and fertility in the males.

DISCUSSION

The first question to be considered is whether the data and analyses here described afford substantial evidence for the existence of a relationship between fertility and body dimensions. Since 41 separate analyses

were performed on each sex—a total of 82 analyses—it might be expected that for some of the characters the *F*-values would approach the 5% level of significance even if there were no relationship between the characters and fertility. In fact, if all of the traits were independent of one another, the *F*-values should reach the 5% level of significance for about 5% of the analyses. Actually, many of the traits are highly correlated with one another, and it is therefore impossible rigorously to consider the question without obtaining all of the cross products among the traits. Nevertheless, it is the view of the authors that the number of significant *F*-values far exceeds the number likely to be obtained by chance, even allowing for correlations among the variables. It does not appear likely that chance alone is responsible for the fact that among the males 28 of the 32 direct measurements have a higher mean value in the above average than in the below average fertility group. Among the females, on the other hand, where only 18 of the 32 direct measurements have a higher mean value in the more fertile than in the less fertile group, chance could be more reasonably invoked. But even among the females the number of differences which exceed the 5% level of significance is greater than would be expected by chance alone. The authors conclude, therefore, that there exists a real difference in certain body measurements between the two fertility groups of both sexes.

The above conclusion is strengthened by the fact that the observed differences fit a definite pattern. For both males and females the measures of body breadth and thickness, namely, weight, biacromial breadth, maximum forearm circumference, maximum midarm circumference, hand breadth, bicondylar breadth of arm, minimum wrist circumference, bicondylar breadth of leg, maximum calf circumference, and minimum ankle circumference, were in every case greater in the more fertile than in the less fertile group. Moreover, the probabilities for the *F*-values associated with these characters and fertility were in every case other than biacromial breadth of females lower than 0.25.

On the other hand, for the measures of length of the body and extremities, namely, stature, cervical height, sitting height, span, forearm length, hand length, middle finger length, and foot length, a rather different situation is evident. Among the females the mean value of the less fertile group exceeded that for the more fertile group for all of the above traits other than hand length, middle finger length, and foot length. However, the probabilities associated with the *F*-values relating these traits with fertility are less than 0.25 only for stature, sitting height, and cervical height; they are greater than 0.50 for span, forearm

length, hand length, middle finger length, and foot length. Among the males, the mean value of the more fertile group exceeded that of the less fertile group for all of the above mentioned 8 measures of length except stature and forearm length. But for half of these traits the probabilties for the F-values relating the trait with fertility exceed 0.50, so that collectively there is little evidence in the males for either a consistent positive or negative relationship between fertility and these various measures of length. It should be noted, however, that for stature in males the interaction between age and fertility was significant at the 1% level. This suggests that there may actually be a relationship between stature and fertility in the males, even though the nature of the relationship varies from age group to age group.

Taken as a whole the data suggest that for the population sampled in this study individuals of both sexes who are above average in fertility tend to be more stoutly built than individuals of below average fertility, that females who are above average in fertility tend to be shorter than females who are below average in fertility, and that there is little consistent relationship between fertility and the various measures of length in the males.

Definite trends are also to be found for the measurements of the face and head. The 6 measures: head length, head breadth, head height, minimum frontal breadth, bizygomatic breadth, and bigonial breadth, are all correlated with general head size. For all of these traits the mean value of the more fertile males exceeded that for the less fertile males. And the probabilities associated with the F-values relating these traits with fertility are in every case less than 0.25. It appears that the males of the Ann Arbor population who are above average in fertility tend to have larger heads than those who are below average in fertility. No such trend is evident in the females. However, there is a relationship among the females between fertility and both the absolute and relative length of the nose, the characters nose height and $\frac{\text{nose height}}{\text{total facial height}}$ tending to be smaller and $\frac{\text{nose breadth}}{\text{nose height}}$ larger in the above average than in the below average fertility groups. These various head and facial characters seem to be involved in popular ideas of masculine and feminine beauty, and it is possible that the relationships between these measurements and fertility are at least in part the result of sexual selection.

There is undoubtedly a relationship between the anthropometric measurements considered in this study and the somatotypes of W. H.

Sheldon. The data appear to indicate that the more fertile individuals of the Ann Arbor population tend to be lower in ectomorphy than are the less fertile individuals. However, our data do not enable us to distinguish accurately enough between endomorphy and mesomorphy to compare the relationship with fertility of these two components of body structure.

The interpretation of the interactions between age and fertility warrants some discussion since the existence, for a given trait, of such an interaction suggests that the relationship between that trait and fertility has not been constant over the time covered by the data. There is some tendency for the differences, D (above average fertility minus below average fertility), between the two fertility groups to be different in the 35–49 year old age groups from those of the other age categories. This is particularly noticeable in those traits for which the interaction between age and fertility is significant. Thus, for both weight and maximum forearm circumference in males these differences are negative for the 35–39, 40–44, and 45–49 year old age groups and positive for all other age groups. For maximum midarm circumference of males the difference is negative only for the 40–44 year old group, and for maximum calf circumference of males it is negative only for the 40–44 and 45–49 year old groups. Since these ages are as of 1952-54 the individuals in the 35–49 year old categories would have been between 14 and 30 years of age in 1933, and it is clear that the most vigorous reproductive years of many of the individuals in these age groups coincided at least in part with the economic depression of that period. It is possible that during this depression the usual positive correlation between fertility and stockiness of build in males was temporarily reversed. For the other traits with a significant F-value for the interaction between age and fertility, namely stature and $\frac{\text{nose height}}{\text{total facial height}}$ of males and ear lobe height of females, meaningful patterns are less evident.

It is unlikely, although perhaps possible, that the relationship between fertility and body type observed in this study is due to the inclusion in the sample of members of racial and cultural isolates which differ both in physical characteristics and fertility. The city of Ann Arbor contains relatively few isolates, as compared with larger cities, and only individuals of European descent were included in the sample. Moreover, the geographic stratification of the sampling would tend to prevent any such isolates from exerting an undue effect on the sample. The data appear

to provide substantial evidence for the existence, in the Ann Arbor population, of a real relationship between physical type and fertility.

The evolutionary and genetic implications of these observations depend on the underlying causal connections. The relationship, in both sexes, between fertility and stoutness may well indicate that stockiness of build tends to increase the fertility of an individual. And this could mean that the genetic structure of the population is changing—that the population is evolving in the direction of greater stoutness. On the other hand, since much of the differentiation with reference to stoutness occurs in middle age, it is possible that the observed relationship between fertility and body build is caused by factors such as marital status or socio-economic level independently influencing both body build and fertility, and in this event these relationships would not be of particular evolutionary significance. The question, then, is whether a stocky build in some way results in greater fertility or whether above average fertility—or the factors associated with it—tends to result in a stocky build. There is, of course, no reason to assume that these two alternatives are mutually exclusive. To assess their relative importance, however, it will be necessary to obtain measurements on young adults prior to differentiation with reference to such factors as marital status and socio-economic level and then to correlate these measurements with subsequent reproductive performance. Such a study is now in progress.

The correlation between fertility and size of head in males or between fertility and nose length and body height in females can scarcely be attributed to an effect on these characters of marital status or of socio-economic level, inasmuch as most of the differentiation with reference to these traits occurs prior to marriage. It is known that variation in these traits is largely under genetic control, and hence the observed correlations with fertility would appear to indicate that natural or sexual selection is operative on them.

SUMMARY

The analyses herein described show that for both males and females of the Ann Arbor population there is a tendency for individuals of above average fertility to be more stockily built than individuals of less than average fertility. However, it is unclear from these analyses whether a stocky build tends to result in above average fertility or whether the factors associated with above average fertility—such as marital status and socio-economic level—tend to result in a stocky build. Consequently, the genetic and evolutionary implications of the correlations are in doubt.

The analyses also suggest that the more fertile males tend to have larger heads than the less fertile males and that the more fertile females tend to be shorter in stature and have smaller noses than the less fertile females. It seems likely that these relationships are due to the operation on these traits of either sexual selection or natural selection, or both, and that they are therefore of appreciable genetic and evolutionary importance.

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STUDIES ON THE OCCIPITAL BONE IN AFRICA

IV. COMPONENTS AND CORRELATIONS OF OCCIPITAL CURVATURE IN RELATION TO CRANIAL GROWTH

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IN previous studies in this series, it has been demonstrated that the occipital curvature in two African physical types, Bushman and Negro, falls into two clear-cut phenotypic classes (Tobias 1958a, 1958b). Bushmen have *curvoccipital* and Negroes *planoccipital* crania. It was further demonstrated that, up to a dental age of about six years, both groups have well-curved occipitals, a recognized infantile feature. Thereafter, a progressive flattening sets in, in Negro crania, whereas Bush crania retain the infantile contour to adulthood (Tobias 1958c).

The existence of two such well-defined categories of occipital morphology has thus led to a study of the developmental processes which engendered them. Once such processes have been clarified, the way is opened to an understanding of the genetical bases of occipital curvature, for, generally speaking, genes operate, not on phenotypic entities, but on processes, altering their rate, timing or direction.

It is the purpose of this paper to enquire whether, in terms of the developmental processes leading to the adult occipital curvature, a particular occipital index occurring in two crania may be assumed to connote identity of pattern or process. The question is posed because of the facile assumption, often made in biometrical physical anthropology, that a metrical resemblance necessarily connotes a genetic affinity. This assumption has been questioned recently by le Gros Clark (1955) and Mednick and Washburn (1956), both of whom have pointed out that metrical and statistical comparisons are often made between biological incomparables.

In seeking a biological understanding of what the measurements reveal, I have been guided by the principle that measurements and indices are simply a means of expressing some morphological features more precisely: measurements are no substitute for morphology. In this study, not only has a biometrical analysis been made of the curva-

ture indices of the occipital bone; but an attempt has been made to dissect the biological significance behind the bare measurements.

MATERIAL AND METHODS

The protocols of the African cranial series utilized in these investigations have been set forth fully in the first paper of this series (Tobias 1958a).

Occipital curvature has been assessed by two indices, both based upon the chord ($S'3$) and median sagittal arc ($S3$) between lambda and opisthion. The simpler Chord-Arc Index is derived by expressing $S'3$ as a percentage of $S3$. Pearson's Occipital Index (Oc. I.) expresses the percentage ratio of the radius of curvature of the occipital bone to the chord $S'3$, assuming that the arc $S3$ is an even curve. The following formula has been devised for Pearson's Oc. I.:

$$\text{Oc. I.} = \frac{S3}{S'3}\sqrt{\frac{S3}{24(S3 - S'3)}}.$$

Values for both indices are higher in crania with flat occipitals, and lower in crania with well-curved occipitals.

A full discussion of the history of the two indices is given in the first paper of the series (1958a), whilst the relative usefulness of the two indices will be considered in the last (1958d).

An answer to the queries posed in the introduction to this paper has been sought in several ways: by analysing measureable components of the occipital arc and chord and by directly comparing different skulls with different indices. Thereafter, the correlation between occipital curvature and other metrical features of the cranium is considered and the deduced morphogenetic changes of the occiput are analysed in terms of modern concepts of cranial growth.

An obvious median point dividing the occipital arc is the inion. It has the advantage that it corresponds in position to the internal occipital protuberance, to which attaches the tentorium cerebelli, separating the cerebrum above from the cerebellum below. The identification of inion may, however, occasion considerable difficulty. In this study the point was taken as the meeting-place of the *supreme* nuchal lines and this generally coincided with the common horizontal tangent to the two *superior* nuchal lines. In assessing the point, the writer was especially guided by a discussion on the subject by Tildesley (1920-21, pp. 184-5).

The position of inion having been determined, I measured the chords

and arcs of the occipital bone above and below inion. From these measurements, two simple chord-arc indices were derived (the Occipital Cerebral Index and the Occipital Cerebellar Index of Sauter, 1941-46). Further, using a Hjortsjo Trigonometer, I computed the angle subtended by the lambda-opisthion chord at inion, from a knowledge of the three chords, lambda-inion, inion-opisthion and lambda-opisthion. These operations were carried out on 55 adult male Somali crania in the Duckworth Laboratory, Cambridge University, and on the Bushman and Hottentot crania in the Anatomy Departments at Cambridge and Edinburgh Universities. For comparison, there were available data on Bushmen, Hottentots, Griquas and Swiss (Sauter 1941-46).

Components of the Occipital Curvature.

The mean values of the Occipital Cerebral and Occipital Cerebellar

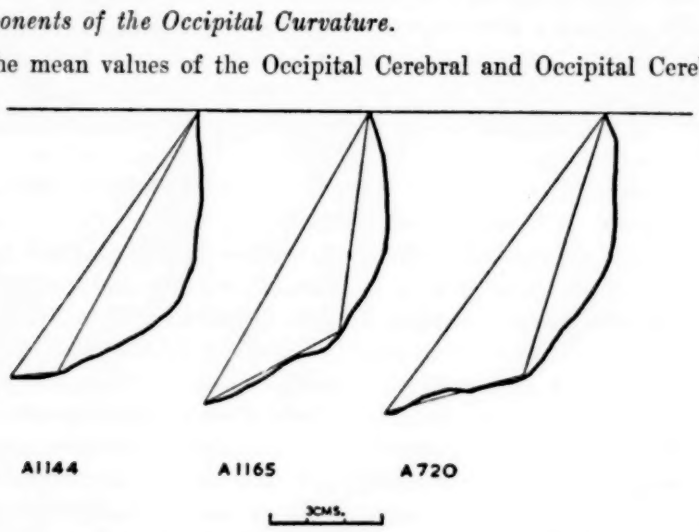

Fig. 1. The Varying Components of the Occipital Curve. In Each Craniogram the Lameda-Inion and Inion-Opisthion Chords have been Indicated. A 1165 and A 720 have Identical Indices and A 1144 a Very Similar One.

A 1144: Immature Bush type with occipital indices 82.8 and 59.42.
A 1165: Bush type with occipital indices 83.5 and 60.15.
A 720: Xosa with occipital indices 83.5 and 60.15.

Indices, and of the lambda-inion-opisthion angle for four African cranial series and one European series are given in table 1.

In all series, the cerebral curvature is more marked than the cerebellar, except that in the Swiss female series, the cerebellar curvature

is slightly greater. The Swiss and Somalis have flatter cerebral curvatures than the Bush, Hottentot and Griqua.

The possibility that the total occipital curvature depended in part on that of the component segments was tested by calculating correlation coefficients (r) between the occipital indices and the indices of the component parts of the occipital bone. The values recorded for these *intraracial* correlations are tabulated below, along with correlation coefficients between the occipital indices and other cranial characters, given by Pearson and Davin (1924) and by Wagner (1937). The italicized values in table 2 indicate a definite correlation, whether positive or negative.

TABLE 1

SERIES	SEX	NUMBER	OC. CEREBRAL I.	OC. CEREBELLAR I.	INION ANGLE
Bushman	M	58	93.7	97.69	125.1°
	F	43	94.0	98.06	120.5°
Griqua	M	14	93.0	94.48	120.8°
	F	2	93.7	100.00	117.2°
Hottentot	M	19	93.0	98.93	118.6°
	F	4	92.8	?	123.5°
Somali	M	55	94.35	98.07	125.1°
Swiss	M	50	96.2	96.46	119.0°
	F	50	96.3	95.22	122.7°

Considering firstly the occipital bone itself, we see that, as was to be expected, both occipital indices ($100S'3/S3$ and Oc. I.) are strongly positively correlated with the angle at inion: a *strong* curvature, in other words, may be expressed as a *low* index or a more *acute* angle, while a *flat* occipital curve is conveyed by a *high* index or a more *obtuse* angle. In exactly the same way, Wagner (1937) demonstrated a very high correlation between Pearson's Oc. I. and the simple chord-arc index (*vide* the first three entries in table 2) . No such high correlation exists, however, with the curvature above or below the inion, as determined by the lambda-inion chord-arc index (occipital cerebral index) and the inion-opisthion chord-arc index (occipital cerebellar index) respectively. There is virtually no correlation with the curvature of lambda-inion, but there is a small though definite positive correlation (.201, .213) with the curvature between inion and opisthion. Up to a point, the more curved the cerebellar part of the occipital arc is, the more curved will the occipital arc as a whole be.

TABLE 2

Correlation coefficients between occipital indices and other cranial characters

	100 S'3/S3	Oc. I.	Series & Sex	Reference
Pearson's Oc. I.	+.963	—	Australian♂	Wagner, 1937
	+.873	—	Sandwich Is.♂	"
	+.868	—	Maori♂	"
Oc. Cerebral Index	+.082	+.071	Somali♂	This study
Oc. Cerebellar Index	+.201	+.213	Somali♂	This study
Lambda-inion-opisthion Angle	+.808	+833	Somali♂	This study
Cranial capacity	—	—.0547	XXVI-XXXth Dyn. Egyptians♂	Pearson and
	—	—.1044	" ♀	Davin, 1924
Max. Cranial Length	—	—.3236	" ♂	"
	—	—.3720	" ♀	"
Max. Cranial (Bi-parietal)	—	—.0882	" ♂	"
Breadth	—	—.1033	" ♀	"
Auricular Height	—	+.0577	" ♂	"
	—	+.0881	" ♀	"
Basion-Nasion Length	—	+.0113	" ♂	"
	—	—.0118	" ♀	"
Sagittal (Nasion-Opisthion)	—	—.2257	" ♂	"
Arc	—	—.2218	" ♀	"
Parietal chord-arc Index	—.252	—	Australian♂	Wagner, 1937
	—.312	—	Sandwich Is.♂	"
	—.221	—	Maori♂	"
Frontal chord-arc Index	+.157	—	Australian♂	"
	+.092	—	Sandwich Is.♂	"
	+.317	—	Maori♂	"
Breadth-Length Index	+.262	+.295	Australian♂	"
	+.464	+.553	Sandwich Is.♂	"
	+.137	+.207	Maori♂	"
Profile Angle (between	—	—.0416	Egyptian♂	Pearson and
standard horizontal plane	—	—.1153	" ♀	Davin, 1924
and nasio-alveolar length)				
Foramen magnum Index	—	—.0317	" ♂	"
	—	—.0311	" ♀	"

This particular correlation is an example of a correlation between two indices, of which a component of one (*S*3) "covers" a component of the other (inion-opisthion arc). Pearson and Davin (1924) showed that correlation is obviously high between "covering" measurements, such as the sagittal arc and the lambda-opisthion arc (*S*3) ($r = .5273$)

or the horizontal circumference and maximal cranial length ($r = .8834$). As they put it (p. 349)

> **The "common cause groups" in all these cases are so obvious that we can state *a priori* that there will be high correlations. They are not true organic correlations, but to a large extent result from what we may term mathematical necessity, i. e. from the compound character of the anthropometric measurements.**

When *indices* are considered, correlations are not significant unless two components "cover" each other in whole or in part, according to Pearson and Davin. Thus, there is a correlation between Nasal and Upper Facial Indices of —.4607(♂) and —.4902(♀): for Upper Facial Height "covers" Nasal Height and Facial Breadth "covers" Nasal Breadth. Where only one component of the first index "covers" one component of the second, the correlation is smaller: for example, the Palatal and Upper Facial Indices are correlated to the extent of —.1325(♂) and —.1743(♀), since Facial Breadth "covers" Palatal Breadth. In other words, the correlation between indices comprising "covering" components increases with the extent of the "covering"; until in the extreme case, when one considers indices with a common component, the correlation is very high.

The occipital curvature being much more highly correlated with the Cerebellar than with the Cerebral Index, it might be expected from Pearson's conclusions that there is more "covering" between one or both components of the Occipital Indices and of the Cerebellar Index than between those of the Occipital Indices and the Cerebral Index. The total occipital arc "covers" absolutely the lambda-inion and inion-opisthion arcs, but the latter comprises the smaller moiety of the total occipital arc (Fig. 1). Thus, Sauter quoted the following mean values for the cerebral and cerebellar arcs:

	MEAN CEREBRAL ARC	MEAN CEREBELLAR ARC
Bush (58♂)	66.1	47.6
Bush (43♀)	61.9	46.4
Hottentots (19♂)	64.2	46.8
Griquas (14♂)	66.0	47.1
Swiss (50♂)	62.8	50.8
Swiss (50♀)	58.8	50.2

It might have been deduced from this relation that the upper or Cerebral Index would be more highly correlated with the Occipital Index, since there is more "covering" of the cerebral arc. Instead, this index is

much less correlated. An examination of the other component of the Cerebral and Cerebellar Indices suggests the probable answer: the chord in each instance is, of course, also "covered" by the total arc (*S*3), but the lower chord approaches much more closely to the size of its arc than does the upper; this is reflected in the higher Cerebellar than Cerebral Indices. The more closely the cerebellar chord approaches the cerebellar arc, *i. e.*, the nearer the Cerebellar Index approaches to 100%, the more highly correlated would the chord be with the total arc which absolutely "covers" the cerebellar arc. The converse is true in the cerebellar region of the occipital bone. Thus, there is a greater degree of "covering" in the cerebellar region and consequently we should expect a higher correlation coefficient between the Occipital and Cerebellar Indices. Of the biological significance of such a correlation, Pearson and Davin (1924, p. 356) have this to say:

> It seems idle to speak of any such correlations as "organic." There are no, or at best very small "organic" correlations between the shapes of different portions of the skull. The observed correlations are due to the existence of common or covering factors in the indices. And notwithstanding the criticisms which have been raised against the term "spurious correlation," it seems to be the appropriate word to describe these correlations which are due, or largely due, to "covering" factors.

It may be mentioned that the size of the component parts of the occipital bone is decidedly more variable than the total dimensions of the bone. Thus, Morant (1930) showed that the lambda-inion and inion-opisthion arcs are more variable than the total lambda-opisthion arc. In other words, the position of inion is most variable. A corollary of this observation is that two cranial series with the same mean occipital index do not necessarily show the same mean curvature above and below inion. For instance, Early Dynastic Egyptian crania and Slovene crania have a mean Oc. I. of 62.0 and 62.1 respectively; yet, if we subtract the Occipital Cerebellar from the Occipital Cerebral Index, we derive values of —3.4 and +2.1 respectively. Skull series with the same overall curvature may have a stronger curvature above or below inion. Again, we find Serbo-Croatian crania with an Oc. I. of 62.8 and a difference between upper and lower curvatures of +0.8; Greek crania with 62.9 and —1.7; and Telei crania of the Solomon Islands with 63.0 and +3.5. Groups possessing the same occipital index may not possess close affinities in detailed structure.

The same is true even of individual crania within a single racial

group. Thus, among my Somali group, crania 2 and 8 both had an Oc. I. of 60.15; yet the Cerebral minus the Cerebellar Index was 90–96.59 (—6.59) and 96.06–95.08 (+0.98). Again crania 22 and 58 had an Oc. I. of 60.31 and differences of the component curves of 96.12–96.33 (—0.21) and 94.78–96.15 (—1.37). Among Bush crania, skulls 1758 and 3731 in the Cambridge Anatomy Department have 59.22 as Oc. I. and 92.9–95.8 (—2.9) and 91.2–98.7 (—7.5) respectively; skull C1369 from an old Hottentot grave at Kakamas, Orange River, has 59.22 as well, but 92.4–95.9 (—3.5). In figures 1, 4 and 5 are shown occipital contours of groups of crania with similar or identical overall occipital indices, but varying from one another in level of inion and other features of their sculpturing.

There is therefore both intra- and inter-racial variability in the relative position of inion and in the relative curvature above and below inion. It is of interest to note that, in the pig, the growth pattern differs between the superior and inferior halves of the occipital bone. Mednick and Washburn (1956, p. 179) comment: "This differential growth potential of the two portions of the occiput . . . is present before birth and is probably genetic in origin."

In short, we see that the occipital indices, while giving an overall picture of the amount of bulging in the occipital region, do not provide any information about the detailed morphology of the occipital bone.

Correlation Between Occipital Curvature and Other Cranial Characters.

There is a rather strong negative correlation between Oc. I. and cranial length (table 2), that is, the longer the skull, the greater the occipital curvature, or vice versa. Clearly this is a "covering" correlation; for part of the very length of a long skull must be due to the occipital prominence, whereas, with a flattish occipital, the skull would seem to have been cut off behind and would be short. Only a single organic process, and not two correlated organic processes, need be postulated. For instance, one genetic process, conditioning a non-expansion or an abrupt posterior termination of the parieto-occipital region of the brain and of its osteogenic and later osseous coverings, would necessarily affect both occipital curvature and cranial length, not to mention a host of other "covering" measurements and indices. One such "covering" measurement is the nasion-opisthion arc, between which and Oc. I. there is a negative correlation of —.2257 and —.2218. Clearly a more bulging occipital (*i. e.*, a lower index) would increase the nasion-opisthion arc.

A strong positive correlation exists between the occipital indices and the cranial (breadth-length) index. A flat occipital (*i. e.*, a high index) makes for a shorter skull, but might leave the cranial breadth unaffected or slightly increased (see below): hence it is not at all surprising to find planoccipital crania tending towards brachycrany.[1] That the growth processes producing occipital curvature are not the only ones involved in the development of brachycrany is clear from the wide difference in *r* between three Australasian cranial series: from .137 in Maoris to .464 in Sandwich Islanders. It must be assumed that other developmental factors are operating to vary the extent to which the cranial index depends on occipital curvature. No amount of occipital flattening would compensate for the lengthening and dolichocephalizing effect of an enormous protruding glabella. Hence, to the extent that other processes are defining the lengthening or shortening, the broadening or narrowing, of a cranium, to that extent will the dependence of, and correlation between, cranial index and occipital curvature be lessened.

An interesting series of correlation coefficients is that between occipital, parietal and frontal curvature. There is a rather strong negative correlation with the parietal curvature. "If the occipital bone is strongly curved, the parietal bone will be relatively flat, and vice versa" (Wagner 1937, p. 72). This is a correlation between two indices which include no common or "covering" components, a type of correlation which, according to Pearson and Davin, should be low. This particular coefficient was not calculated by Pearson and Davin, else they might have recognized it as an exception. They would have found further, though slighter, exceptions in the degree of correlation between the occipital and frontal indices, and still less marked correlations between the frontal and parietal (—.085, —.143, —.145, Wagner, 1937, p. 72). Here, in the parietal and occipital regions are indices without covering components which seemingly break Pearson's rule that "There is no correlation between the shapes of individual parts of the skull."

[1] Since this paper was written, I have received from Professor Mario Cappieri a copy of a paper (Cappieri, 1957), in which he discusses *inter alia* brachycephalization. He stresses that the tendency towards brachycephalization is accompanied by a tendency of the angle between the two parts of the occipital squama, superior and inferior, to become progressively more obtuse, *i. e.*, for the skull to become what we have here called planoccipital. This occipital change is part of a process whereby modern skulls tend towards a rounded form, consequent upon (a) restriction of the base and (b) extension of the calvaria and walls. Cappieri says: "In this process of extension it appears that the parietal bones have the task of conforming to the dynamics of the bones articulated with the base."

Wagner pondered over this correlation between parietal and occipital curvature: he demonstrated that the parietal chord-arc index was negatively correlated with the cranial (breadth-length) index ($r = -.271$ in Australian males, $-.375$ in Sandwich Island males and $+.005$ in Maori males). On the other hand, the occipital index was positively correlated with the cranial index. Since both indices were correlated

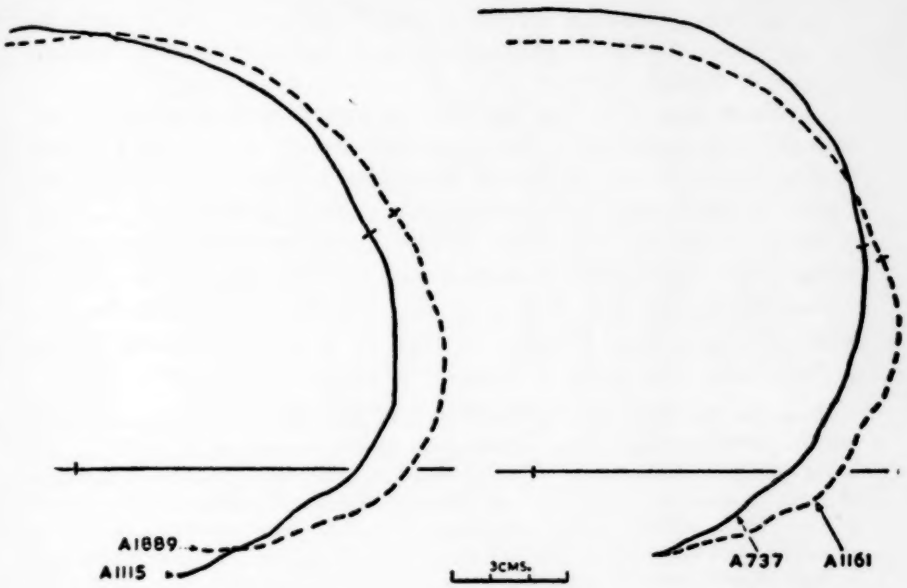

FIG. 2. PARIETO-OCCIPITAL CONTOURS OF PLANOCCIPITAL AND CURVOCCIPITAL CRANIA. NOTE THE INVERSE RELATION BETWEEN PARIETAL AND OCCIPITAL CURVES IN EACH PAIR OF CRANIA.

A 1889:	Xosa cranium with occipital indices		75.0 and 54.4
A 1115:	Bush type cranium with	" "	86.3 and 63.9
A 1161:	Bush type cranium with	" "	74.9 and 54.4
A 737:	Zulu cranium with	" "	93.1 and 84.0.

with head shape, they must be correlated with each other. However, Wagner then goes on to say that "the flattened occipital bone and more curved parietal bone of the Sandwich Islanders and Bainings (of New Britain) are considered to be a *consequence* of their high breadth-length index." (italics mine).

It is almost redundant to point out that the breadth-length index is not a primarily genetic feature: what is inherited is a growth potential

in various directions. It has already been stated above that a single change in the growth pattern of the parieto-occipital region would be enough to account for both planoccipital and brachycranial indices, neither index being the *consequence* of the other. When one considers, in addition, the parietal index, it is not immediately apparent that the *same change* in the parieto-occipital region would bring about high curvature in the parietal region, unless there were an overall genetic or ontogenetic pattern for the entire cranial vault, such that changes in one region of the vault would necessarily be accompanied by compensatory changes in another region.

Evidence is not lacking for such an overall pattern within which regional and compensatory variations may occur. When the occipital bone is larger, the parietal bones are smaller (r for bregma-lambda arc ($S2$) and lambda-opisthion arc ($S3$) is —.1587 in males and —.3422 in females—Pearson and Davin, 1924). In other words, although the overall size of parietal and occipital may not vary greatly, the position of lambda between them does (Fig. 3). This observation is in keeping with the more general statement derived by Pearson and Davin (1924, p. 345) from their study of variation in cranial dimensions:

> It seems very noteworthy that the least variable characters of the skull are not the individual bones, but combinations of these which go to make up the broad structure of the skull. There is, we think, little doubt that the variability of measurements on the individual bones of the skull—anatomical as distinguished from anthropometric measurements—must show considerably higher variation than measurements on its structure as a whole. In other words two individuals with closely corresponding anthropometric measurements might have widely different anatomical measurements. The end product might be nearly the same although produced by component cranial bones growing in different proportions.

They go on to deny that this result connotes an important organic law controlling growth; instead they demonstrated algebraically that, from the theory of variation and the definition of the coefficient of variation itself, it can be deduced that "the percentage variation of a character which is the sum of a number of biologically varying units not highly correlated together, will be very considerably less—roughly as the inverse square root of the number of units—than the mean percentage variation of the units themselves. Thus it comes about that the chief anthropometric characters of head and skull have a markedly less variability than the anatomical characters, by which we understand measurements confined to a single bone."

This algebraic proof is perfectly satisfactory when applied to the variation of a *series* of skulls. It does not, however, explain what happens in an individual skull during its growth. The mechanism by which one pattern of occipital growth is associated compensatorily with another pattern of parietal growth is not explicable in algebraic or statistical terms: it must be regarded as a developmental, not a statistical

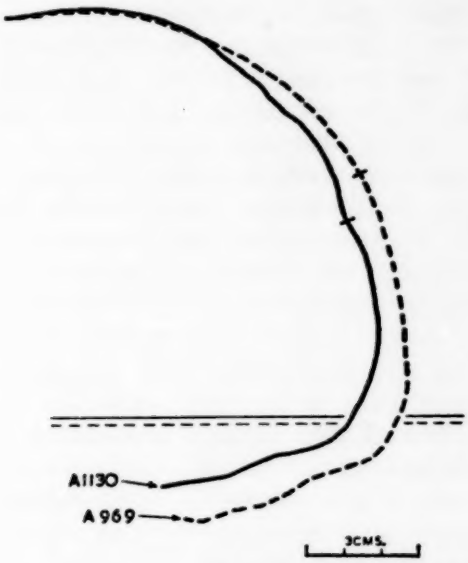

FIG. 3. TWO SKULLS WITH SAME OCCIPITAL INDICES SHOWING VARYING LEVEL OF LAMBDA. THE CRANIOGRAMS ARE ORIENTED IN THE FRANKFURT HORIZONTAL WITH BREGMA COINCIDING.

A 1130: Bush type with occipital indices 78.8 and 56.22.
A 969: Zulu with occipital indices 78.7 and 56.22.

phenomenon, and we cannot escape the conclusion that the correlations do suggest "an important organic law controlling growth." Perhaps Wagner (1937, p. 72) came close to the truth when he suggested in a foot-note:

> This negative correlation (between occipital and parietal indices) gives an interesting insight into the etiology of the curvature. Pearson and Davin . . . regard it as "probable that the Occipital Index is a rough measure of the development of the cerebellum" (p. 332). The above correlations, and even more the correlations between the breadth-length and occipital chord-arc indices and Pearson's Occipital Index make it seem likely that it is the cranial shape as a whole

which is principally responsible for the curvature of the occipital bone. This does not mean that the underlying brain does not play a part in determining the form and size of the cranium. One must reckon with an intimate interdependence of local and general factors acting partly on the brain, partly on the cranium.

His last reference to the brain arises presumably from Pearson's view "that heredity of family or race provides the brain shape, brain size and brain detail, that the function of the cranial bones is to cover this brain, and that their organic correlations are produced in growing to its soft parts. . . . Notwithstanding its plastic character the brain moulds the head shape and size. . . ." Pearson probably oversimplified the position by attributing an exclusive influence to the brain, whereas Wagner, whilst accepting the general influence of the brain, was prepared to countenance direct effects on the cranium, such as those of the temporal and nuchal muscles later demonstrated by Washburn (1947).

A number of "developmental complexes" (Tobias, 1955) must be instrumental in moulding the lineaments of the skull, among which are at least the following: brain-size and form; tooth morphology, size and arrangement; the manner and degree of chewing and the development of the masticatory muscles associated with the nature of the diet; posture, which might influence the neck muscles, the nuchal plane and the occipital bone generally; the degree of development of the para-nasal sinuses; pedomorphic or gerontomorphic tendencies (or the influence of retarding or accelerating mutations and environmental factors); other more specific morphological features probably of primary genetic determination, *e.g.* prominence of brow-ridges, angulation and position of cheek-bones, "overshooting" or "undershooting" of nasal bones (Birdsell, 1951), tori palatinus et mandibularis.

Nevertheless, whatever genetic and other variants may be introduced in the morphogenesis of the skull, the correlations between parts suggest some integrating mechanism affecting the vault as a whole. The manner in which this integrating mechanism operates may become clearer after we have considered the implications for occipital curvature of modern concepts of cranial growth.

Occipital Morphology and Cranial Growth.

Greater or lesser expansion of the supra-occipital, especially the planum occipitale, at the lambdoid suture, must play its part in moulding the curvature of the occipital: for if the upward expansion of the supra-occipital "stops short," then the adequate covering of the parieto-

occipital lobes of the brain can be effected only by a downward curve of the parietal bone, especially its hinder part. This is simply a part of the explanation of what our indices have already revealed: the less curved the occipital, the more curved the parietal. If it were assumed that a certain parieto-occipital curvature is necessary to cover the parietal and occipital lobes of the cerebrum, as well as the cerebellum, it would follow that a part of this total curvature is contributed by the occipital bone and a part by the parietal bone. If rather more curvature were contributed by the occipital, rather less would be contributed by the parietal; and vice versa. Now if the planum occipitale of the supra-occipital expanded above only slightly, there would be correspondingly less opportunity for the occipital to contribute to the total parieto-occipital curve. Hence, the "shifting" position of lambda would itself have consequences for the occipital curvature. This line of reasoning presupposes a certain pattern of growth along the lambdoid suture, namely one in which accretive growth may be unequal on the two faces of the suture. Such a pattern is precisely what studies on skull growth have revealed.

The recent change in orientation towards cranial growth and especially towards the role of the sutures has been reviewed by Baer (1954), Moss (1954) and Mednick and Washburn (1956). Moss (1954, p. 334) has summarised the two main schools of thought about the role of the suture as a site of growth:

> One group views the suture as a primary site of growth of the skull, functioning as an "epiphyseal center" (Mair, 1926; Weinmann and Sicher, 1947). Another group regards the suture as only a passive mechanism allowing for relative movement between the bones of the skull during growth (Mijsberg, 1932; Brash, 1934). In this view, the periosteal tissues are the primary growth sites.

Massler and Schour (1951, p. 99) demonstrated by vital staining of the rat skull with alizarin red "S" that growth of the skull vault "occurs primarily by a rapid deposition of bone at the approximating margins of the bones which comprise the various cranial sutures." This was confirmed for the rat by Baer (1954) and for the pig by Mednick and Washburn (1956). Moss (1954) meantime used a variety of extirpative techniques to elucidate further the influence of sutural tissues on the growth and shape of the rat calvaria. He was able to conclude:

> The shape of the calvarial bones is not predetermined by the location of the sutures. It is possible to produce overgrowth of calvarial bones by many methods with consequent displacement of sutural position. This phenomenon appears to

depend on the period of development of the osseous tissue. Calvarial sutures mark the plane of articulation between adjacent bones. The location of the sutures is determined by the relative growth of the adjacent bones.

This is simply another way of saying what Massler (1941—quoted by Baer 1954) had demonstrated much earlier, namely that the amount of growth is not always equal on the two sides of a suture. Massler had shown that the parietals (in the rat) sustain a greater increment of growth than the interparietal at the suture between them.

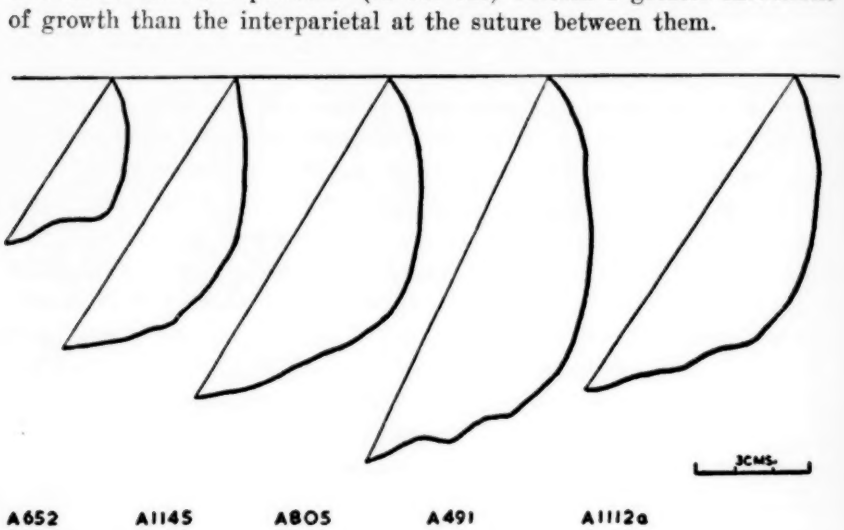

FIG. 4. VARIATIONS OF OCCIPITAL ARCHITECTURE ASSOCIATED WITH SIMILAR OCCIPITAL INDICES. ALL CRANIOGRAMS ARE ORIENTED ON THE FRANKFURT HORIZONTAL.

A 652: Nyasa infant with occipital indices 80.3 and 57.3
A 1145: Immature Bush type with " " 80.0 and 57.05
A 805: Xosa child (ca. 5 years) " " 80.25 and 57.2
A 491: Zulu male with occipital indices 80.1 and 57.2
A 1112a: Bush type male with " " 80.2 and 57.2

On such a conception of skull growth, it is easy to understand the relatively "shifting" position of lambda with the consequences of this shift for occipital size and curvature. Furthermore, Baer (1954) has demonstrated that increase in the height of the occipital bone results from incremental growth at the exoccipital-supraoccipital junctions and at the supraoccipital-interparietal suture. The exoccipital elements begin to unite with the supraoccipital at approximately 45 days, and fusion is completed by 60 days of age, when the occipital complex comprises a

single bone. Subsequent increase in height of the occiput results from the slow, continued deposition of bone at the superior margin of the supraoccipital until 100 days of age.

Working on the pig, Mednick and Washburn (1956) showed that in the infant pig (1–8 weeks), sutures are active sites of skull growth, "accounting for the primary extension of the bones of the vault." Later, secondary appositional growth on both ecto- and endo-cranial surfaces, though mostly on the former, is responsible for increasing the thickness of the bones, while secondary resorption occurs between the two tables (not on the endocranial surface), to create the great layer of diploic and sinus spaces in the pig. In a thick-skulled form like the pig, the outer table departs very radically from the architecture of the inner table, so that, while the inner table remains closely moulded to the form of the brain, the ectocranial contour diverges from the brain form most markedly. In thin-skulled forms, such as man and the rat, the outer table closely conforms to the pattern of the inner table, and this in turn to the surface of the brain. Appositional growth in such a thin-skulled form as man is minimal; according to Mednick and Washburn (1956), "this results in the rounded contours of the infant skull persisting throughout the life of man." Therefore, sutural growth is predominant in thin-skulled forms, while apposition and resorption are relatively unimportant in man. It might be inferred from their conclusion that apposition is correspondingly more important in forms of man with heavy bony excrescences like orbital and occipital tori, *e.g.*, palaeolithic man and Australian aborigines: the difference between Rhodesian Man with his heavy occipital torus and a Bush skull with very similar occipital index (A 1161) is at once apparent (Fig. 5). Here, the same occipital index is apparently the result of two different processes: firstly, in Rhodesian Man, heavy appositional growth (probably with a well-curved occipital) and, secondly, in A 1161, marked curvature of the occipital, moulded by sutural growth mechanisms to the underlying brain.

Baer's work has thrown light on another aspect of the problem, the genesis of occipital architecture in general. He has shown that there are certain sites of bone deposition and resorption which are "concerned primarily" with change in the *shape* and *proportions* of the skull, rather than with *dimensional* increase of individual bones. Skull form depends closely on the specific sites, timing and direction of growth. The details of the attainment by the rat of adult occipital conformation need not detain us here (see Baer, 1954, pp. 113-4), but his analysis gives an

insight into the morphogenetic processes involved in the curving of the occipital bone. Like the human child's occipital bone, the bones of the immature rat's vault are highly curved: during development a marked flattening of the dorsum of the calvaria occurs (much as the occipital bone of Negroes flattens out with maturation). Weinmann and Sicher (1947) and Brash (1934) believed that the flattening of the arc of each bone is effected by differential apposition and resorption on the outer and

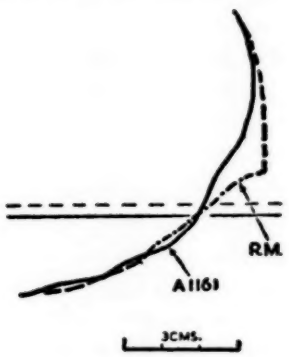

FIG. 5. EXTREME VARIATION OF OCCIPITAL CONTOUR ASSOCIATED WITH SIMILAR OCCIPITAL INDICES. BOTH OCCIPITALS ARE ORIENTED ON THE FRANKFURT HORIZONTAL WITH LAMBDA COINCIDING.

R. M.: Rhodesian Man with occipital indices 75.17?? and 54.5??
A 1161: Bush type with occipital indices 74.9 and 54.40.
The portion of R. M. shown by a dot-and-dash line has been reconstructed.

inner surfaces of the vault; but Massler and Schour (1951) and Moss (1954) have pointed out that this endo- and ecto-cranial osteogenesis occurs in the rat calvaria *under 60 days of age*. Sutural growth is simply one part—in a thin cranium, the main part—of periosteal osteogenesis early in development and it enables the bones, which are "floating outwards" (Moss) in the neuro-cranial capsule on the expanding brain, to expand into the presumptive sutural areas. Baer, however, found insufficient evidence of differential apposition and resorption to account for the radical changes in relationship between the cranial bones in the flattening and other alterations of contour, which occur *during the first 100 days of life*. He therefore postulates that the changes in form of the rat skull are the result of *spatial re-orientation of the individual bones*. There are, as it were, hinge-movements between the bones; but hinge-movements of curved bones would make for bumps and disharmony

in the general contour of the skull. A harmonizing process occurs. The differential growth of the inner and outer cortical plates observed by Baer at the sutural margins represents the mechanism of adjusting the changing relationship of adjoining bones.

Some such process may well account for the changing pattern of parieto-occipital curvature in man. Ford (1956), working on the fetal human skull, has demonstrated that the parts of the occipital bone do indeed change their spatial orientation towards one another during fetal life. The posterior part of the cranial base undergoes a flattening to compensate for its slower rate of growth, as compared with the anterior part of the base and with the brain and vault. This flattening is achieved by changes in the spatial orientation of the parts of the cranial base and the occipital bone. The angle between the septal and basi-occipital parts of the base opens out from a value of 104.9° at 10 weeks to 122.4° at 40 weeks; likewise the angle between the basiocciput and the foramen magnum (limited posteriorly by the inferior margin of the supraoccipital) increases from 109.0° at 10 weeks to 135.0° at 40 weeks. Finally, the supra-occipital moves to a more horizontal position relative to the cranial base.

The result of these processes is to produce the increasing occipital fullness which is particularly characteristic of man. It may be added, here, that it is these processes which result in the occipital curvature coming into being. For, if the antero-inferior part of the supraoccipital is, as it were, "tucked in" horizontally, the upper part must curve round to meet the parietal bone, in order to close the vault posteriorly. This would account for the marked occipital curvature of infants of different races and it is adequately explained by the type of mechanism postulated by Baer.

We have already seen that the parieto-occipital contour remains relatively unchanged with post-natal growth in the Bushman and related groups. Our measurements have shown that this occipital fullness develops, too, in Negroid infants, but with post-natal growth, is subsequently largely flattened out. If Baer's explanation were to apply to these changes as well, one would expect the occipital bone to undergo a hinging movement downwards about the lambdoid suture; this would be accompanied by differential ecto- and endo-cranial accretion and resorption across the lambdoid suture to smooth the skull contour. Such a downward swing would be feasible only if the underlying brain were meantime undergoing alterations in shape, such that the upper parieto-occipital fullness were becoming reduced, perhaps by a lateralizing

tendency. Such a lateralizing tendency would at once account for both the planoccipital contour and the meso- to brachycrany which usually accompanies it. A glance at adult planoccipital crania, however, serves to remind us that the downward hinging of the hinder pole of the skull does not in fact commence at the lambdoid suture. Instead it begins about mid-way between bregma and lambda, so that the posterior part of the parietal bone is itself angulated on the anterior part, to face almost directly backwards (Skull A 737, Fig. 2). This would suggest that the hinging movement at lambda would tend to create so marked a disharmony between parietal and occipital bones as to require, on the parietal face of the lambdoid suture, a large amount of endocranial osteogenesis and ecto-cranial resorption to maintain the smoothness of the general brain-covering.

If there is a lateralizing tendency of the brain, it must then extend over the parietal as well as the occipital lobes of the brain.

Functional Significance of Varied Occipital Morphology.

Pearson always regarded the aim of the Occipital Index as being "to provide some crude measure of cerebellar development" (Pearson and Davin, 1924). In fact, he went so far as to claim that "Among modern man the Negroes stand highest in index and lowest in motor-coördinating capacity." While the latter claim would certainly not stand the scrutiny of modern researches, the whole basis of such reasoning must be questioned. When we are considering different races of man, a big cerebellum does not necessarily imply better co-ordinating capacity. Even if it did, however, it is not at all clear that with a big occipital curve we are *in fact* dealing with a big cerebellum. We are dealing with a total parieto-occipito-cerebral *and* cerebellar conformation: with the relative degree of development and with the shape of the posterior part of the cerebrum and of the cerebellum; with the degree to which the occipital pole overhangs the cerebellum. In a planoccipital skull, the parietal lobe turns sharply downwards just in front of obelion; in a curvoccipital cranium, there is a much slighter deflection from further back in the parietal bone. It does not follow, however, that the parietal lobe housed in a curvoccipital vault is any bigger than that in a planoccipital calvaria. For one thing, the relatively broader vault of a planoccipital skull permits more of the parietal lobes to be housed laterally, as if to compensate for the deficiency of space posteriorly. We can conceive of a re-distribution of parietal lobe material to fit several

different surface contours, *e. g.*, those of scaphocephaly and oxycephaly. The change in shape, *per se*, is no more likely to affect the functioning of the parietal lobe than is a variety of sizes within human limits of variation. Likewise, a cerebellum protruding posteriorly is likely to be no more efficient than one well-covered by the occipital lobe.

It is clear that we must abandon once and for all any notion that the occipital index provides a guide to the degree of cerebellar development and, *a fortiori*, to the capacity for motor co-ordination.

We can more confidently assert that the occipital index reflects the degree to which the infantile parieto-occipito-cerebral and cerebellar contour has been retained or departed from, during the post-natal morphogenetic changes in the brain. As to the functional significance of a down-curved parietal lobe and flat occipito-cerebellar contour, in contrast with a slightly curved parietal lobe and well-curved occipito-cerebellar surface, this type of study can throw no light on functional differences of these regions of the brain among various human races.

General Conclusions on Biological Significance of Occipital Curvature.

1. The total occipital curvature is more closely correlated with the Occipital Cerebellar Index than with the Occipital Cerebral Index, presumably because there is more "covering" of the components of the cerebellar index by the components of the occipital index.

2. Within any cranial series, the more planoccipital crania are more likely to tend towards brachycrany.

3. The flatter the occipital, the more well-curved the parietal bone, and vice versa.

4. Both intra- and inter-racial variability exists in the relative position of inion and in the relative curvature above and below inion. Groups of crania and even individual crania of the same series, though possessing the same occipital index, may not possess close affinities in these details of their morphology.

5. The occipital index gives an overall picture of the amount of bulging in the occipital region; it does not provide any information about the detailed structure of the occipital bone.

6. It is suggested that the following growth processes are involved in the establishment of the occipital curvature of the adult human being:

(a) The initial fetal curvature is established by the flattening of the

cranial base: as Ford showed, this occurs by hinging movements opening the basi-cranial angles in front of and behind the basi-occiput and between the supra-occipital and the foramen magnum.

(b) Ossification in the brain capsule above the nearly horizontal anterior part of the supra-occipital results in the occipital curving upwards and forwards towards the lambdoid suture.

(c) Accretion of bone occurs on both sides of the lambdoid suture, but the relative amount of ossification on the two sides varies. Hence the position of lambda is variable: the planum occipitale may in consequence be relatively expanded or unexpanded. The curvature of the upper part of the occipital will in turn vary; an expanded planum occipitale is likely to result in more of the general parieto-occipital curve being taken up by the occipital and less by the parietal. If the planum occipitale is small, less of the parieto-occipital curve will be taken up by the occipital and more by the parietal.

(d) Our correlation coefficients have, however, shown that the curvature of the upper part of the occipital (from lambda to inion) is less correlated with the total occipital curve than is the curvature of the lower occipital arc (from inion to opisthion). Some other factor than a shortening and flattening of the lambda-inion arc must therefore be sought to explain the flattening of the occipital arc which occurs in some races.

(e) It is suggested that with post-natal growth, and especially from about the time the permanent teeth begin to erupt, there occurs a re-arrangment of the brain substance of the parietal and occipital lobes. This rearrangement would seem to comprise a relative flattening and lateralizing of these lobes. Such changes are accompanied by spatial re-orientation of the parietal and occipital bones.

(f) The occipital bones hinge downwards at the lambdoid suture; they accomplish this movement without actively compressing the brain. In fact, they probably follow the afore-mentioned brain re-ararngements passively, if the results of all the cited experimental studies on cranial growth apply to man; and there is no reason to doubt that these general principles, though not necessarily the details, are so applicable.

(g) The hinge-movements referred to in (f) tend to create irregularities of contour along the lambdoid suture: these are compensated for by differential ecto- and endo-cranial osteogenesis along the

juxtaposed margins of the parietal and occipital bones. The degree to which the parietal bone is involved in the consequent downward angulation is inversely related to the amount of expansion of the supraoccipital along its lambdoid margin; a well-expanded supraoccipital will permit much of the parieto-occipital curve to be taken up by the occipital and only a small amount of the curve will be contributed by the posterior part of the parietal bone. A very flat occipital will result when much of the parieto-occipital downward deflection is in the parietal bone itself. The degree to which the parietal bone turns downward is governed by a proximate and a remote mechanism: more immediately, it depends on the amount of endocranial osteogenesis and ectocranial resorption along the lambdoid margin of the partietal; more remotely, the whole cranial contour must be presumed to be related to the conformation assumed by the underlying brain (Wagner, 1935, 1937).

(h) The hinge-movement, it is suggested, accounts for the very marked reduction in the tempo of cranial length increase which Goldstein (1936) found at this age. At the same time, it is of interest to note that the width increment, both maximum cephalic and minimum frontal, was reduced after five years of age *by a much smaller amount* than were the height and length diameters. It is tempting to attribute this to the lateralizing tendency of the brain and to relate to this phenomenon as well a brachycephalization from about 5 or 6 years of age.

(i) The persistence of the infantile parieto-occipital brain contour into adulthood in some races (*e.g.*, Bushman, Hottentot), and the loss of this contour by parieto-occipital flattening and lateralization, of a *moderate* degree in some human types (*e.g.*, Eastern Bantu and Eastern Hamites) and of a *marked* degree in others (*e.g.*, Western Bantu, Southern Bantu and True Negro), are the main conclusions drawn from a study of the occipital indices of different modern African racial series.

7. It is concluded that the occipital index provides no guide to the degree of cerebellar development and, *a fortiori*, to the capacity for motor co-ordination.

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SOCIAL DISTANCE IN EPIDEMIOLOGY

AGE OF SUSCEPTIBLE SIBLINGS AS THE DETERMINING FACTOR IN HOUSEHOLD INFECTIVITY OF MEASLES

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CERTAIN contagious diseases of man maintain their reservoir of infection solely by a continuous chain of contact between infective and susceptible persons. This is particularly true of infections of the upper respiratory tract that are transmitted through expiration, coughing and sneezing. Many of these viruses and pathogens have short persistence outside the human body and it is, therefore, evident that a successful chain of infection must depend to a large degree on how close the susceptible persons are to the infective ones.

It has long been realized that geographical distance is an important determinant in the spread of contagious disease. The instances of isolated communities that for decades have escaped invasion of common infections, such as measles, and where such invasions when they finally occur take exorbitant epidemic proportions, belong to the classical knowledge in epidemiology. It is also recognized that even in dense urban communities the spread of such diseases may assume a peculiar pattern of movement that confines epidemics of the disease to certain sociologically defined areas in the larger community (*e.g.*, Meyers, 1949). The social contacts of a susceptible person and the kinds of people with whom he is in daily intimate contact are epidemiologically more decisive than the population density of the area in which he lives.

The concept of social distance, although recognized as an epidemiologic principle, has not been used in the exact terminology adapted by sociologists who by meticulous interviewing determine the number of a person's social contacts. Yet, how much simpler would it be to use the attack rates of contagious disease as a unique measure of social contacts between groups than to use a number of graded questionnaire items.

The reverse assumption, however, has long been a cornerstone of the study of the infectivity of diseases. Chapin (1925) established the simple device of *secondary household attack rates* as a measurement of maximum infectivity of the contagious disease. The attack rate is computed as: (total number of cases in household minus primary cases) divided by (total number of susceptibles in the household minus primary cases). The underlying assumption is that susceptible persons living in the same household are at maximum human contact—or at a minimum social distance—and, furthermore, the degree of contact is assumed to be the same in all households in a given epidemiologic area. This measurement has been highly informative in practical appreciations of the difference in infectivity of various contagious diseases. In particular, it has been a useful measurement in field trials of biologic products such as the passive immunization with gamma globulin against infectious hepatitis (Ashley, 1954) and active immunization against whooping cough (Med. Res. Council, 1956). Because of the high expected attack rates at household exposure, fewer observations are needed to obtain significant rate difference than would be necessary in total community attack rates.

However, there are several limitations in the use of secondary attack rates either as a measure of the maximum infectivity of disease or the social structure of households. In order to arrive at the attack rate, one must be able to determine the susceptibility of the individuals *a priori*. In certain instances, this can be done fairly accurately through the medical history, provided one deals with infections that practically always result in frank disease. Measles and whooping cough are some of the few examples. Other infections that can confer immunity without disease leave the troublesome procedure of measuring antibodies before the household is infected, which limits the study to special circumstances. Retrospectively, in the case of measles, susceptibility can be determined fairly satisfactorily. The only exception concerns infants that, for a certain length of their first year, have passive protection from transplacental antibodies.

Another limitation occurs when several susceptibles in one household are exposed to one or more primary infectives with short periods of infections. The possibilities exist that the subsequent cases among these susceptibles can be caused either by the primary case or occur as a chain where the secondary cases give rise to teritary cases among susceptibles that escaped infection from the primary case but were infected by the secondary case. Such chains will result in a higher secondary attack

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rate in large households than in smaller households, and the secondary attack rate defined by Chapin therefore becomes a function of the social structure of the community, with higher secondary attack rates in communities with larger family units.

Efforts to correct the secondary attack rate for family size were initiated by Greenwood (1931) who formulated his chain theory as one of the first attempts to introduce the stochastic process in epidemiology. Greenwood's theory is most easily understood in the case of a family with three susceptibles where one becomes a primary case that is infected from the outside. Four kinds of happenings are now possible in respect to infection or escape of the two remaining susceptibles if "p" is the "true" secondary attack rate, and $p = 1 - q$

(0.0)	Two escapes	Probability	q^2
(1.0)	One secondary case	"	$2pq^2$
(2.0)	Two secondary cases	"	p^2
(1.1)	One secondary, one tertiary	"	$2p^2q$

The probabilities refer to the expected frequency with which families with these happenings would occur. Obviously, the sum of the probabilities is unity. The secondary attack rate as defined by Chapin would be

$$\tfrac{1}{2}(2pq^2 + 2p^2 + 4p^2q) = p(1 + pq).$$

Since the term $(1 + pq)$ is larger than unit, the secondary attack rate in families with 3 susceptibles would be larger than that in families of 2 susceptibles, where one would expect a rate of p.

Greenwood was able to confirm this theory on actual observations of Stocks and Karn during measles epidemics in St. Pancras, London (1926). However, the data were collected with the limitation that families were listed by number of cases subseqent to the primary case only, so that the categories (2.0) and (1.1) were indistinguishable. Thus, the observed number of families with no, one, and two subsequent cases were respectively 120, 93 and 86. Solving for p in $p + p^2 - p^3 = (93 + 172)/598 = 44.3$ and computing the expected numbers, he obtained *122.4*, *88.2*, and *88.4*, which is a satisfactory agreement.

However, the Greenwood chain theory received a severe blow, when later Wilson *et al.* (1939) published observations on measles collected from 1928 to 1932 in Chapin's former office site, Providence, Rhode Island. This imposing monograph contains among other valuable data a chapter ("Greenwood's brat and other matters") where the chain

theory is tested against data where the categories (2.0) and (1.1) were separated. The distinction was made by careful recording of the time interval between cases. Those families where the two secondary cases occurred with more than 8 days' interval (the incubation period of measles) between onset of typical symptoms were listed as (1.1)—one secondary and one tertiary case—while an interval of less than 8 days placed the family in category (2.0)—two secondary cases.

Actual observations were:

	FREQUENCY OF FAMILIES	
	Observed	Expected *
(0.0)	51	51.2
(1.0)	67	66.5
(2.0)	262	175.3
(1.1)	36	123.0
	416.	416.0

* based on $p = 0.649$.

It is readily seen that the expectancy in category (1.1) exceeds the observation far more than random errors would permit. Several explanations were offered, of which one published in one of Greenwood's last papers (1949) seems likely. He abandons the idea of a uniform milieu in all families of a community and proposes the infectivity (p) of measles is distributed in a certain way, depending on the family characteristics.

Assume, for example, the simple situation that the community consists of two equally sized groups of families, in one of which the infectivity is 0.50 and in the other the infectivity is 1.0. The frequency of the four categories of families—(0.0), (0.1), (2.0), and (1.1)— would then be 0.25, 0.25, 0.25, and 0.25 in the first group, and 0.0, 0.0, 1.0, and 0.0 in the second group. The total community would then present a frequency of 0.125, 0.125, 0.625, and 0.125. An observer who is unaware of the difference in families, and hence computes the overall infectivity from the latter distribution, finds $p = 0.6621$ and computes the expected distribution in the respective categories as 0.114, 0.151, 0.439, and 0.296, which in most cases would be acceptable with the first two categories but quite unacceptable for the last two categories—the families with two subseqent cases.

The characteristic deviation of that observed data from the chain theory can also be expressed in the fact that the risk of secondary cases always appears higher than the risk of tertiary cases. This phenomena

is also found in the case of 4-child families with one primary case. The quaternary attack rates that are occasionally found in these families tend to be higher than the tertiary rate, but with doubtful significance (Abbey, 1952).

Bailey (1953) and later in his monograph (1957) analyzes in detail the idea, suggested by Greenwood, of a variation in the infectivity rate among the household. He found that by assuming a simple distribution of p among the households (Beta-distribution) the data observed in Providence could be fitted for both 3-child and 4-child families with one primary case. The distribution of infectivity was statistically the same in both sets, J-shaped with infinite ordinate at $p = 1$.

Thus, the observed data are compatible with the assumption that circumstances in various households differ to the extent that the risk of a susceptible child contracting measles from its infective sibling depends on environmental factors of the household. It is inviting to speculate on the nature of these factors: size of dwelling, cleanliness, cultural habits, etc., none of which was specified in the observed data.

However, there are data in the Wilson report which will be treated in the following and which can be shown to explain satisfactorily and simply the nature of difference in household infectivity. It rests solely with the age composition of the children, and the clue is contained in Wilson's careful tabulations of the events in the 2-child families.

A re-analysis of the infectivity in 2-child families with one primary case.

Tables 38a, b, c, d, and e in Wilson's reports present 1340 2-child families with one primary case. In 500 of these families the second child was reported to have had measles previous to the primary case. Among the remaining 840 families were 70 families in which the exposed child was less than one year of age. Twenty five of these 70 children contracted measles subsequent to the primary case, giving an attack rate of 0.36 which is considerably lower than the rate in the 770 families with susceptible, exposed children over one year old; 621 of these 770 children became infected ($p = 0.807$). Since it is entirely possible that an unknown proportion of the 70 infants was protected by maternal antibodies, it is necessary to exclude this group from the following analysis of age factors responsible for infection risk, excluding active and passive immunity.

Wilson's tables 38c, d, and e presented the families by age of primary case and age of secondary case (c) of susceptible escape (d), and of

immune sibling (e). The data of the former two tables can be arranged in a single table containing the attack rate among susceptible siblings by age of the primary case and by age of the exposed susceptible. While the overall attack rate is $621/770 = 0.807$, inspection of the table shows that there is an obvious dependence of the attack rate both with the age of the susceptible and with the age of the primary case.

Preliminary polynomial analysis.

In order to obtain an estimate of the nature of the dependence of attack rate on age, a polynomial regression analysis was performed with attack rates as the dependent variable on components of age of the primary case (x) and age of the susceptible exposed (z).

TABLE 1

Matrix of normalized sums of products for the computation of co-fficients in Equation (1)

	x	x^2	z	z^2	xz	y	
x	4,876					39.24	
x^2	71,672	1,207,038				—157.48	
z	2,395	42,630	8,547			—177.38	
z^2	35,783	684,833	125,194	2,067,504		—3,468.61	
xz	50,817	885,698	79,762	1,236,195	1,057,824	—1,153.12	
y						43.0778	(135)
					Variance of components	11.2921	(5)

Coefficients	standard error
$b_x + .06078$	.0206
$b_{xx} - .00498$	.0017
$b_z + .00155$	.0185
$b_{zz} - .00398$	.0011
$b_{xz} + .00470$	.0019
b_0 0.6212	.0196

The coefficients in the following equation were determined:

$$y = b_0 + b_x x + b_{xx} x^2 + b_z z + b_{zz} z^2 + b_{xz} xz \qquad (1)$$

where y is the expected attack rate.

The matrix of sums of products and sums of squares is given in table 1, together with the coefficients of the variables and their standard error, derived from solving the five equations.

$$S_{iy} = \Sigma\, b_{ij} \cdot S_{ij}$$

where $S_{ij} = \Sigma i \cdot j - (\Sigma i \cdot \Sigma j)/770$; i and j stand for x, x^2, z, z^2, and xz, and S_{ij} are given in the matrix.

The order of magnitude of the coefficients and their confidence limits permit a simplified prediction, as given in equation (2)

$$y = 0.8958 - .00105(x + z - 15)^2 - .00306(x - z - 4)^2 \qquad (2)$$

This equation has almost the same information as that of equation (1). The variance components due to regressions are 11.1819 and 11.2921, respectively. Equation (2) has two essential variables $x + z$, the sum of ages of the two siblings, and the difference in their ages (primary minus the secondary). The latter term provides 85% of the information but the 15% due to the sum of ages is statistically significant.

In table 2 the data from Wilson's tables are rearranged by these two variables, and the fraction of secondary cases over primary cases is given for each combination. The prediction equation (2) consists of two parabolic planes. However, when the sums and difference are large the predicted attack rate attains negative values.

A more natural, but biometrically less manageable, expression is the following:

$$y = 0.8979e^{-0.001(x+z-15)^2 - 0.005(x-z-4)^2} \qquad (3)$$

which was obtained by iterative procedures. This equation never gives negative values of y. On the graphs of figures 1 and 2 the observed frequencies are plotted by sum of ages and difference of ages, respectively. The lines are drawn with corrections, according to equation (3) for $(x - z)$ in figure 1 and $(x + z)$ in figure 2.

The marginal totals of table 2 are used to plot the points in the graphs.

While the agreements seem acceptable there is need for a test of goodness of fit. Most of the individual fractions in table 2 represent too small numbers for the direct application of the Chi-square test. For such purpose the table was condensed to give reasonably high numbers as in table 3.

The expected numbers of secondary cases were computed by adding the expectancies within each of the 15 groups according to equation (2) and to equation (3). The Chi-square test was now performed for each of the two sets of summed expectancies with the following results:

Equation (2)	$\chi^2 = 6.726$	with 10 degrees of freedom
Equation (3)	$\chi^2 = 6.722$	

TABLE 2

Secondary cases over secondary susceptibles by age of the two siblings

TABLE 2

Secondary cases over secondary susceptibles by age of the two siblings
age of primary case—age of susceptible sibling
(x — z)

Sum of Ages x + z	—18	—17	—14	—11	—8	—7	—6	—5	—4	—3	—2	—1	0	1	2	3	4	5	6	7	8	9	10	13	Total
1												0/1													0/1
2											3/4														3/4
3												2/4		5/6											7/10
4									1/1		0/1		1/1		8/9										10/12
5										2/5		2/2		3/4		10/13									17/24
6									0/2		3/3				15/15		6/9								24/29
7								1/1		5/7		2/4		1/4		18/21		17/19							44/56
8							0/2		1/1		6/6				24/27		20/25		11/15						62/76
9								2/2		3/4		4/5		10/10		26/32		19/21		4/5					68/79
10							0/1		2/2		3/6				35/42		19/24		7/7		3/4				69/86
11						0/1		0/1		3/3		5/5		18/20		33/41		11/12		2/4		1/1			73/88
12									2/2		7/7				21/24		17/19		2/2		1/2		1/1		51/57
13				0/1				1/1		6/6		2/3		7/8		22/22		4/4		1/2					43/47
14							0/1		1/2		5/9		5/5		15/16		11/11		1/2						38/46
15								2/2		4/5		2/5		8/8		8/8		2/3		2/3					28/34
16							1/2		2/2		4/5				8/8		3/5		2/2				2/2		22/26
17								2/4		4/7		1/2		2/2		5/5		1/1							15/21
18					0/1		0/1		4/4		4/6		0/1		5/5										13/18
19						0/2				1/1		5/5		2/2		3/3									11/13
20							1/1		2/3		3/5														6/9
21		0/1						0/1		1/2		1/1		1/1											3/6
22	0/1		0/1										1/1								1/1				2/4
23										1/1				1/1										0/1	2/3
24									1/1		2/3														3/4
25						0/1				1/2															1/3
26					0/1				0/1																0/2
27										1/1		0/1													1/2
28					0/1																				0/1
29						0/1														0/1					0/2
30											1/1				1/1										2/2
33								1/1						1/1				1/1							3/3
35														0/1											0/1
36															0/1										0/1
Totals	**0/1**	**0/1**	**0/1**	**0/1**	**0/3**	**0/5**	**2/8**	**9/13**	**16/21**	**32/44**	**41/56**	**26/38**	**7/8**	**59/68**	**132/148**	**125/145**	**76/93**	**55/61**	**23/28**	**9/15**	**5/7**	**1/1**	**3/3**	**0/1**	**621/770**

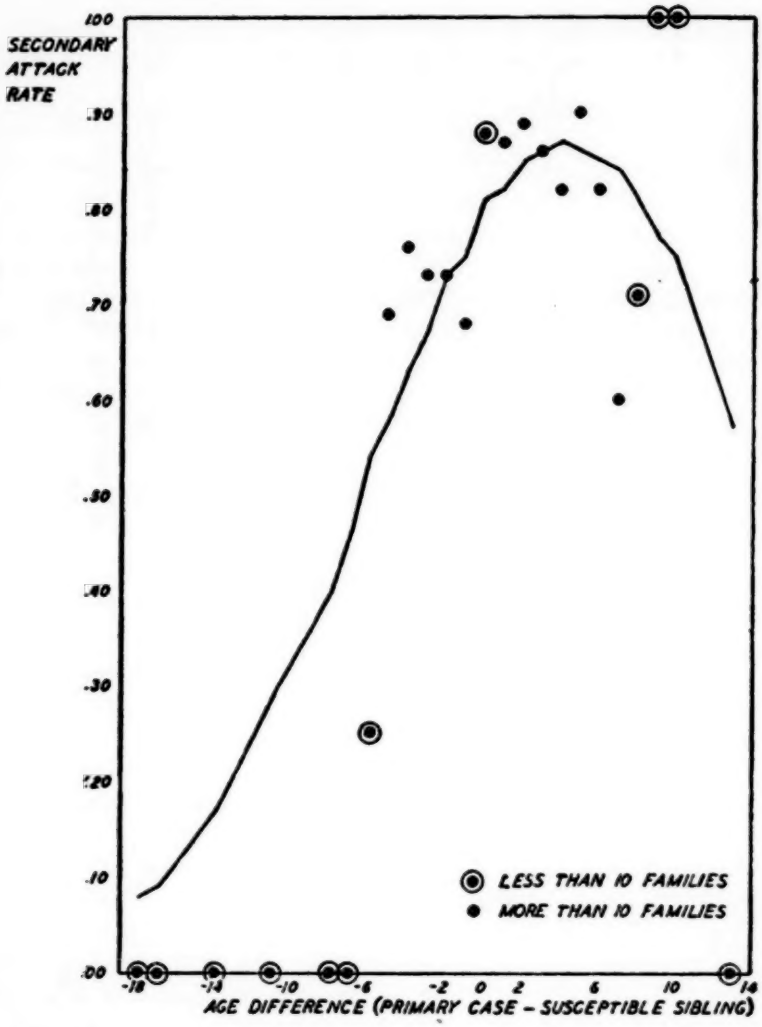

FIG. 1. ATTACK RATES AMONG SUSCEPTIBLE SIBLINGS IN 2-CHILD FAMILIES WITH ONE PRIMARY CASE, BY AGE DIFFERENCE BETWEEN THE TWO CHILDREN. THE CURVE IS DRAWN ACCORDING TO EXPECTANCY FROM EQUATION (3) ADJUSTED FOR THE SUM OF AGES FOR EACH AGE DIFFERENCE.

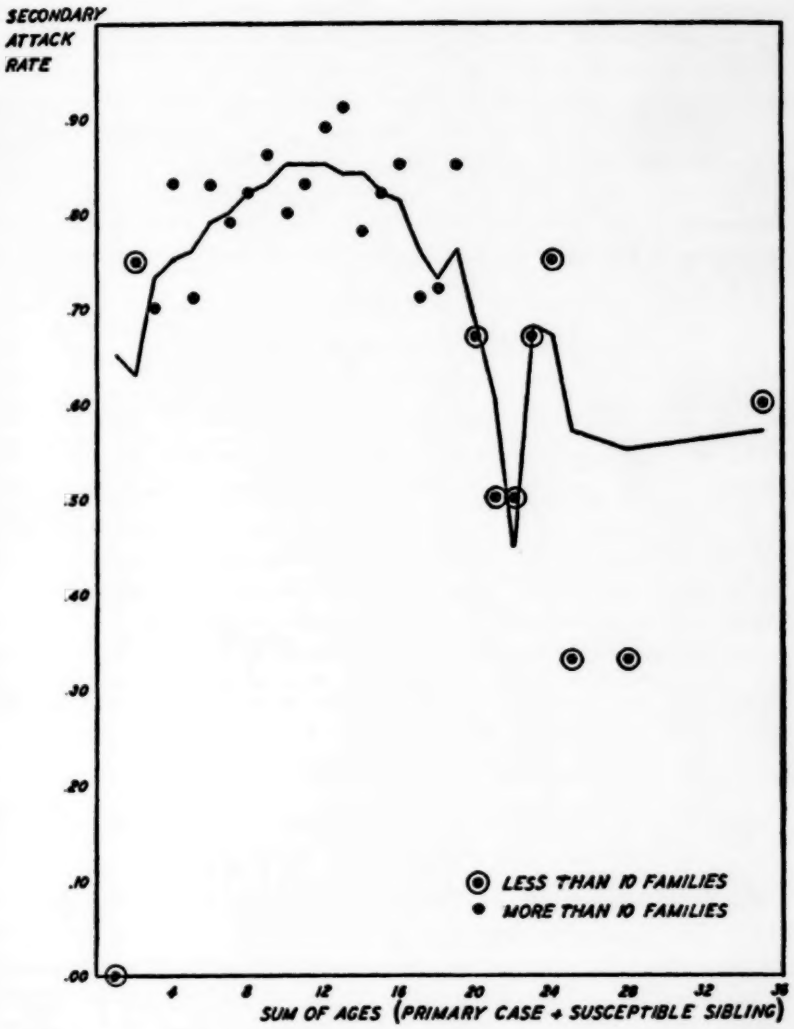

FIG. 2. ATTACK RATES AMONG SUSCEPTIBLE SIBLINGS IN 2-CHILD FAMILIES WITH ONE PRIMARY CASE, BY SUM OF AGES OF THE TWO CHILDREN. THE CURVE IS DRAWN ACCORDING TO EXPECTANCY FROM EQUATION (3) ADJUSTED FOR AGE DIFFERENCE FOR EACH SUM OF AGES.

The minimum Chi-squares obtainable, if the coefficient of $(x+z-15)^2$ is assumed equal to zero, are:

	Minimum Chi-square	Loss of information disregarding $(x+z-15)^2$	
Equation (2)	15.278	8.552	with 2 d.f.
Equation (3)	14.836	8.114	

Because of the significant increase in Chi-square ($p = 0.02$) caused by omission of the sum of ages, this component is assumed to be of real

TABLE 3

Aggregated attack rate from table 2
$(x-z-4)^2$

$(x+z-15)^2$	484–25	16–4	1–0	Total
441–100	14/29	19/25	11/14	44/68
81–49	22/34	54/64	61/74	137/172
36–25	22/34	60/69	64/77	146/180
16–4	49/65	61/72	96/107	206/244
1–0	23/35	41/44	24/27	88/106
Total	130/197	235/274	256/299	621/770

influence on the attack rates although less important than the age difference. The Chi-square value of table 3 omitting both components assumes the highly significant value of 48.11 (14 d.f).

Application of age difference as socio-epidemiological distance to 3 and 4-child families by a simple Monte Carlo method.

The foregoing analysis of infectivity of measles in 2-child households permits the conclusion that the ages of the children are important determinants of the chance of infection. Indeed, in the present material the variation in infectivity among various households is sufficiently explained from the age values in that deviations from the suggested prediction equations are of the order of random variation.

Thus, the variation in household infectivity that was postulated on a theoretical basis by Greenwood, and proven mathematically feasible by Bailey, now seems to lend itself to individual measurement through a simple recording of the age pattern of the susceptible children. Unfortunately, no records have been published of the age distribution of

children in 3 and 4-child families in which measles occurred, to the extent of those of 2-child families.

The average ages of the children in the 3-child families are given in Wilson's report but these means seem to include immune children in the families. Table 45 gives the number of families in which the oldest, middle, or youngest child, respectively, was the primary case. The specification is somewhat incomplete but the following information can be extracted, concerning families with three susceptible children:

	PRIMARY CASE		
	Oldest	Middle	Youngest
No. of families	274	110	29
No. with no secondary case	28	16	8
Percent	10.2	14.5	27.5

The chance of escape is highest when the youngest child is the primary case which is in agreement with the terms $(x-z-4)^2$ in equations (2) and (3).

In order to test the actually observed distribution of infection chains in 3 and 4-child families on the basis of age differences between children, it would be necessary to know the exact distribution of the ages in such families and insert the probabilities for each chain which could be derived from the predictions obtained in the 2-child families. This would constitute a rather laborious task, preferably to be computed on a modern electronic machine.

As a substitution for such involved computations, we have tried to approach the problem through a simple card game that has been "played" by students at the Harvard School of Public Health during the last four years* The accumulated outcome of this game is compared to the actual observations on measles in Providence.

Description of card game.

The "players" are divided into groups of three and four, symbolizing families of susceptible children of those sizes. Each player prepares a deck of bridge cards so that he has a distribution of values as follows:

Value	Ace	2	3	4	5	6	7	8	9	10	J	Q	K	
Number	1	1	2	4	4	4	4	3	3	2	1	0	1	Total 30

* The author gratefully acknowledges the enthusiastic and meticulous participation of the students in Course Epidemiology 7d from 1954-1957.

The value of the cards symbolizes the age of a child and the distribution is roughly that of the age distribution in susceptible 2-child families. The game is played so that there is only one primary case. One of the players is elected to be the primary case. He turns a card at random, the value of which is his age (x). Each other member now turns a card from his deck, showing his age. All those whose ages are within the range $x + m$ to $x - m$ are secondary cases. The distance (m) can be fixed at 3, 4, or 5, the greater the distance the higher the infectivity. It turns out that $m = 4$ will give results rather close to the infectivity found in 2-child families.

Tertiary cases will be found among those players who were outside the range $x + m$ to $x - m$ but might be inside a range $z + m$ to $z - m$ where z is the age of any secondary case. In the 4-child families one can continue the chain by searching for quaternary cases among those who had escaped becoming tertiary.

Example:

4-child family $m = 4$

	AGE	RESULT
Primary	3	primary case
Sibling A	10	tertiary
Sibling B	7	secondary
Sibling C	1	secondary

B and C are secondary cases because they are within the range of $3 - 4$ to $3 + 4$. A is a secondary escape but a tertiary case because he is within the "infective range" of Sibling B.

In this manner the principle of the social distance has been simplified to an all or none phenomenon while the theory assumes a decreasing chance of infection with increased age distance. The asymmetry that was found in the 2-child families symbolized by the term $(x - z - 4)^2$ is abandoned and the notion of the sum of the ages has also been discarded. In spite of these rough approximations the distributions obtained by these card games are strikingly similar to those found by actual observation (table 4).

As mentioned previously, the disparity of the measles observations from the Greenwood chain theory is essentially expressed in the fact that the chance of becoming a secondary case (sec. attack rate) is higher than the chance of becoming a tertiary case (tert. attack rate). As seen

TABLE 4

Comparison of family distribution by chain type

Observed (Measles, R. I. 1928-32) and experimental data (Monte Carlo method)

A. Families of three

CHAIN TYPE	OBSERVED (0–22 yrs.)	EXPERIMENTAL $m = 4$	
1, 0, – (a)	49	42	
1, 1, 0 (b)	44	67	
1, 1, 1 (c)	58	60	
1, 2, – (d)	368	251	
Total (N)	519	420	
	sec. rate 0.807	0.749	Formula: (b+c+2d)/2N
	tert. rate 0.569	0.438	" c/(b+ c)

B. Families of four

CHAIN TYPE	OBSERVED 7 mos.–10 yrs.	EXPERIMENTAL $m = 4$	OBSERVED 0–22 yrs.	EXPERIMENTAL $m = 3$
1, 0, –, – (a)	4	18	10	28
1, 1, 0, – (b)	3	24	7	40
1, 1, 1, 0 (c)	1	3	3	7
1, 2, 0, – (d)	8	31	36	71
1, 1, 1, 1 (e)	4	5	4	13
1, 1, 2, – (f)	3	25	3	10
1, 2, 1, – (g)	10	86	13	61
1, 3, –, – (h)	67	204	75	109
Total (N)	100	396	151	339
sec. rate	0.827	0.760	0.750	0.650
tert. rate				
(1 exposure)	0.500	0.509	0.382	0.286
(2 exposures)	0.556	0.735	0.265	0.462
quat. rate	0.800	0.625	0.571	0.650

Formulas:

sec. (b+c+2d+e+f+2g+3h)/3N
tert. 1 (c+e+2f)/2(b+c+e+f)
tert. 2 g/(d+g)
quat. e/(c+e)

in table 3, the 519 3-child families observed in Providence have a computed secondary attack rate of 0.807 with a tertiary attack rate of 0.569. 420 "games" played in groups of three show the same general distribution, although they differ significantly by the chi-square test. However, the "experimental" families show deviations in secondary and tertiary attack rates that are similar to those of the observed measles data.

Similarly, the pattern of secondary, tertiary, and quaternary attack rates in 4-child families is the same in the observed and experimental series, the general tendency being a high secondary attack rate, a lower tertiary, and a higher quatenary. The tertiary attack rate is here separated in the two situations where the escapes can be exposed to either one or two infectives.

Two values of m—"the epidemiological distance"—were tried in the experiments in order to show the influence of an over-all change of the infectivity on the pattern of the three attack rates.

Two hypotheses are then available to explain the discrepancy between observed data and the chain theory: the hypothesis of age as the determining factor, and the hypothesis of the infectivity with undefined family milieu as suggested by Greenwood and Bailey. The essential difference is that the first assumes different infection risks *within the same family*, determined by the number of social contacts that exist between children of various ages. The latter assumes that the same infection risk exists for all members of a given family but that this risk varies between families and, presumably, Greenwood's chain theory would hold within a given family but disagree when several families of a community are aggregated.

By means of the expectancy of risk that was derived from the 2-child family (equation 3) one can compute the probability of events (chain types) in a 3-child or 4-child family with given age distribution.

Example. Given 3 children: A—2 years, B—7 years, and C—12 years. Equation (3) gives the following infection risks:

Probability	of	A	infected	when	B	infective	0.86
"	"	B	"	"	A	"	0.73
"	"	A	"	"	C	"	0.75
"	"	C	"	"	A	"	0.34
"	"	B	"	"	C	"	0.88
"	"	C	"	"	B	"	0.59

Now, assuming that B, the 7 year old, is the primary case, the chain types have the following probabilities:

CHAIN TYPE *	PROBABILITY
1, 0, —	0.06
1, 1, 0	0.25
1, 1, 1	0.18
1, 2, —	0.51
	1.00

The secondary and tertiary attack rates, computed as in table 4, are 0.725 and 0.419 respectively.

Similar computations can be made for a 4-child family with the ages 2, 7, 10, and 12 years. If the 7 year old is the primary case, the probabilities are:

CHAIN TYPE *	PROBABILITY
1, 0, —, —	.017
1, 1, 0, —	.043
1, 1, 1, 0	.021
1, 1, 1, 1	.051
1, 1, 2, —	.055
1, 2, 0, —	.063
1, 2, 1, —	.394
1, 3, —, —	.356
	1.000

with the "attack rates"

secondary	0.717
tertiary (1 exp.)	0.535
tertiary (2 exp.)	0.862
quaternary	0.708

The frequency of the chain types, as computed by this method, changes according to the relative age of the primary case. The overall attack rate is highest when the oldest child is the primary case and lowest when the youngest child is the primary case. In some instances the tertiary attack rate is in fact higher than the secondary attack rate.

DISCUSSION

We have previously used the terms age difference, social distance, and epidemiological distance as almost synonymous, although the relationship between age difference and infection risk was the only demonstrable

* In the formula of the chain type, 0 means exposed susceptible persons were not infected; dash (—) means either that the susceptibles were all exhausted at this point or that the chain of infection was interrupted because there were no infections in the previous period of infectivity.

fact. The concept of social distance is merely introduced as a hypothesis to explain the mechanism behind the association of relative age and risk of infection. Immunological explanations can hardly be made for this phenomenon since we are reasonably certain to be dealing with persons who are equally susceptible to the disease. It is also hard to believe that age would determine the degree with which the person is infective. For that reason we have resorted to a mechanism that is based upon the social inter-relation of siblings of various ages, and we assume that the direct contact between siblings decreases with age difference even if they are living in the same household. Direct proof is of course needed, but considering the enormous amount of work that is involved in collecting purely epidemiological data, one can easily imagine how complicated a simultaneous epidemiological and sociological study would be. So far the idea of social distance between siblings of various ages is merely based on common human experience.

If we turn to a community of children of approximately the same age in which an epidemic occurs, age difference would probably mean very little to determine the epidemiological distance. In a boarding school of boys between 10 and 14, say, the risk of infection would be related to phenomena of social structure, classes, dormitory, and friendship groups within the school. In this connection it is of interest to point to the work of Abbey who analyzed the sequence of attack rates in several epidemics in such schools. The main result of her work is that a uniform infectivity rate could not be accepted but that it was necessary to assume an oscillation of attack rate during an epidemic similar to the one that can be demonstrated in 3 and 4-child families where the secondary attack rate is higher than the tertiary, etc.

Abbey does discuss the possibility of variation in the contacts among individuals and tries a mathematical model where the population has two sub-populations with less contact between than within these sub-groups. She does find a better fit to the observation with this limited set of conditions but concludes, " However, to obtain conclusive evidence a more realistic model of conditions likely to be found in an actual community is needed. For example, one giving each individual several contact rates." Apparently what is needed in such a study is a simple score for each individual that would be just as workable as the actual age of siblings in small families.

SUMMARY

Reanalysis of data on infectiousness of measles in 2-child families leads to a formula for the probability of infection (y) that includes the

age of the primary case (x) and the age of the second, susceptible exposed child (z) as follows:

$$y = 0.8979e^{-0.001(x+z-15)^2-0.005(x-z-4)^2}$$

This expression is in support of a hypothesis that risk of contact infections in households can not be assumed to attain a uniform value, but is dependent on the social inter-relation between children as expressed by their relative age.

This hypothesis explains quantitatively the distribution of infection chains in 3- and 4-child families, that are at odds with the stochastic chain theory assuming a uniform risk of infection. The hypothesis also seems more realistic than the postulate of Greenwood and Bailey which assumes some theoretical distribution of infection risk between families but not within families.

A simple Monte Carlo method, using playing cards, yields distributions similar to those observed in the field.

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THE DEPENDENCE OF PERMANENT TOOTH LOSS UPON TOOTH MORBIDITY

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INTRODUCTION

THE purpose of the present paper is to call attention to a new way of looking at tooth *mortality* (i.e., average number of teeth missing or in need of extraction per examinee). We have adopted the term *lethality* for the tooth mortality per 100 decayed, missing or filled (*DMF*) teeth. Because of marked differences in caries attack rates, standards of treatement and other factors in different populations, we have selected a single example using dental statistics on a single population of Hungarian boys and girls. Comparisons are made only between subgroups of the same population. The chief purpose of this presentation is merely to discuss the usefulness of the concept of tooth lethality in statistical studies of the relationship of loss to dental caries. In the past various statistical aspects of tooth loss have been stuided (Brekhus, 1928, 1929; Knutson and Klein, 1938; Wisan, 1938; Cheyne and Drain, 1940; Munblatt, 1941; Knutson, 1942; Klein, 1943; Hagan and Cook, 1949; among others). In summarizing their findings Finn (1952) states that "loss of the teeth is inversely correlated with the amount of dental treatment received." Mortality of the permanent teeth is therefore recommended as an index of the adequacy and effectiveness of the dental health service rendered to the public. The economic status of the parents was shown to influence the rates of loss of teeth and the ages at which particular teeth are lost. It is also shown that girls have a higher tooth mortality than boys, the difference being a reflection of the higher caries rates among the permanent teeth in girls.

While these statements can be accepted unequivocally, a more thorough discussion of the effects of tooth morbidity upon tooth loss still seems necessary. In our opinion this problem has hitherto not been studied in a fully satisfactory manner since past studies deal only with *tooth mortality*; they correlate loss of teeth only to the number of examinees. In studying the dependence of tooth loss upon the morbidity of teeth it seems a more proper procedure to correlate tooth loss with the number of carious teeth. In this way *tooth lethality* may be computed.

To exemplify this concept we have studied tooth loss data for three urban communities in Hungary (Eger, Gyöngyös and Ózd). Dental caries has been shown to be sex, age and tooth specific (Klein and Palmer, 1941). We have therefore taken these factors into account. The sexes are considered separately. The data are grouped by age; as no differences were demonstrated within age groups if these were grouped by ages 10–11, 12–14 and 16–18, the data are presented in this way. In school age children, only the first permanent molars are lost in sufficient numbers to serve for a statistical analysis. The number of first permanent molars lost is therefore presented for each sex and the various age groups as a ratio of the number of individuals examined and also as a ratio of the number of DMF first molars.

RESULTS

Results for boys and girls in the three age brackets are summarized in table 1. Since the number of DMF first molars varies between 0 and 4, the children in each age group were divided into five subgroups (with 0, 1, 2, 3, or 4 DMF first permanent molars). Tooth mortality and tooth lethality figures are computed for each of the subgroups.

Within the same age bracket, no significant difference is found in the first molar lethality between sexes for children with the same number of DMF first molars. Therefore it seems permissible to unite the data of both sexes to table 2.

In analyzing the data presented in tables 1 and 2, increase of mortality and lethality of the first permanent molar with advancing age is obvious. Since this relation to age is well known from former data in the literature, no further discussion seems necessary.

In comparing the subgroups with increasing numbers of DMF first molars, an increase not only of the mortality but also of the lethality seems obvious although a few data are seemingly contradictory. With increasing numbers of DMF first molars, we find an increase not only in the number of first molars extracted and requiring extraction but also an increase in first molar lethality (number lost per 100 DMF). In examinees with 1 DMF first molar, a lower percentage of this tooth was found to be extracted and needing extraction than in persons with 2, 3, or 4 DMF first molars. A few contradictory findings deserve special attention: in table 1, girls of 16 to 18 years of age with 4 DMF first molars display lower lethality rate than those with 3 DMF first molars, but the difference is not statistically significant. Similarly, an identical

lethality figure is evident in table 2, in the 16 to 18 year olds with 3 and 4 DMF first molars. In boys of 10 to 11 years in table 1, and in the

TABLE 1

Mortality and lethality of first permanent molars in boys and girls of different age brackets

AGE [1]	SEX	NUMBER OF PERSONS	NUMBER OF DMF FIRST MOLARS PER 100 PERSONS	NUMBER OF FIRST MOLARS EXTRACTED AND REQUIRING EXTRACTION PER 100 PERSONS: FIRST MOLAR MORALITY	LOSS OF FIRST MOLARS PER 100 OF DMF FIRST MOLARS: FIRST MOLAR LETHALITY [2]
10–11	Boys	168	0	0	0
		148	100	5	5.0±1.8
		165	200	6	3.0±0.94
		140	300	16	5.0±1.1
		97	400	28	7.0±1.3
	Girls	111	0	0	0
		117	100	2	2.0±0.92
		179	200	5	2.5±0.83
		156	300	18	6.0±1.1
		140	400	48	12.0±1.4
12–14	Boys	214	0	0	0
		197	100	8	8.0±1.9
		249	200	16	8.0±1.2
		185	300	24	8.0±1.2
		200	400	46	11.5±1.12
	Girls	163	0	0	0
		168	100	3	3.0±1.3
		234	200	17	8.5±1.3
		226	300	28	9.0±1.1
		202	400	59	15.0±1.3
16–18	Boys	333	0	0	0
		160	100	26	26.0±3.5
		134	200	68	34.0±2.9
		118	300	122	40.5±2.6
		120	400	178	44.5±2.3
	Girls	205	0	0	0
		191	100	25	25.0±3.13
		192	200	70	35.0±2.43
		149	300	120	40.0±2.31
		167	400	149	37.0±1.9

[1] Age to the nearest birthday.
[2] Computed to the nearest 0.5 unit.

total group of this age bracket in table 2, a lower lethality prevails with 2 than 1 DMF first molar; again, the difference is not statistically significant.

DISCUSSION

In explaining the phenomenon of an increase in tooth lethality with an increase of tooth morbidity within the same age-group of the same population, several items are to be taken into account.

In Hugary all school children receive treatment through the school dental service without charge. If the school dental service would work

TABLE 2

Combined data of both sexes on mortality and lethality of the first permanent molars in different age brackets

AGE [1]	NUMBER OF PERSONS	NUMBER OF DMF FIRST MOLARS PER 100 PERSONS	NUMBER OF FIRST MOLARS EXTRACTED AND REQUIRING EXTRACTION PER 100 PERSONS: FIRST MOLAR MORTALITY	LOSS OF FIRST FIRST MOLARS PER 100 DMF FIRST MOLARS: FIRST MOLAR LETHALITY [2]
10–11	279	0	0	0
	265	100	4	4.0±1.2
	344	200	6	3.0±0.65
	296	300	17	6.0±0.80
	237	400	38	9.5±0.95
12–14	377	0	0	0
	365	100	6	6.0±1.24
	483	200	16	8.0±0.87
	411	300	27	9.0±0.82
	402	400	53	13.0±0.84
16–18	538	0	0	0
	351	100	26	26.0±2.34
	326	200	69	34.5±1.9
	267	300	120	40.0±1.73
	287	400	160	40.0±1.44

[1] Age to the nearest birthday. [2] Computed to the nearest 0.5 unit.

optimally, there should not be any loss of first permanent molars in school age children. It is, however, unavoidable that some children escape dental treatment, being absent when examination is carried out by the school dental surgeon or when treatement is to be performed. Furthermore, primary school is entered only after the 6th birthday is passed. Inasmuch as an acceleration of the eruption of the permanent teeth occurs in children in urban communities (Adler, 1957), it may happen that some children are first examined after an unduly long exposure of

the first permanent molars, by which time decay is so far advanced that instead of restorative treatment extraction is to be resorted to. In individuals whose first permanent molars have erupted some time before dental examinations and treatment are instituted, the more first permanent molars are carious in a person, the greater the chance that one or another tooth cannot be saved. This would be so even if the progress of dental decay is no more rapid in individuals with more carious first molars than in those with less. Clinical experience points, however, to differences in the progress rate of dental decay, a more rapid progress being displayed in persons with less resistance to caries, i.e. having at equal chronological ages more teeth decayed.

Supposing that absence from school at the time of dental examination and/or treatment is due to chance alone (contrary findings are not known to us), a higher percentage of lethality is expected in persons displaying more than one DMF first molars. Our findings in the age brackets 10 to 11 and 12 to 14 are in best agreement with these expectations.

A surprising finding is recorded, however, in the 16 to 18 year olds. In this age bracket the difference in the lethality between examinees with 1 and 4 DMF first molars is considerably reduced as compared with the same difference in the two younger age brackets. In the younger age groups persons with 4 DMF first molars display a lethality figure that is more than double that of persons with 1 DMF first molar, whereas in the 16 to 18 year olds the increase is only 1.5 fold. On the other hand, the increase in the lethality figures is seemingly more marked from 1 DMF first molar to 2 DMF first molars. We are unable to explain this finding satisfactorily although several possibilities have been taken into account.

First of all, different principles of selection were used in the younger and elder age brackets; in the two younger groups only children born and reared in the same urban area were considered, while in the 16 to 18 year olds all pupils attending the secondary schools were counted. It is well known to us that several pupils were attending secondary schools in the three communities who were enrolled in primary schools in rural districts where no school dental service was available. In this way it might have occurred, inasmuch as rural people lay less stress upon preserving posterior teeth, that several first molars were extracted in this group of secondary school children that would have been saved in urban children of equal age. Since caries prevalence is lower in rural than in urban pupils (Adler, 1954) such extractions were supposed to occur mainly in the subgroups with a lower number of DMF first molars.

However, taking into account tooth decay and tooth loss rates of first molars in children of 16 to 18 years born and reared in the three urban communities—as summarized in table 3—, no difference is conspicuous in comparison with the total population of the same age bracket; rather more uniform lethality figures are seen in the restricted group beginning with 2 DMF first molars. So this explanation was discarded.

TABLE 3

Combined data of both sexes on mortality and lethality of first permanent molars in children of 16 to 18 years, born and continuously resident in the three urban communities

NUMBER OF SUBJECTS	NUMBER OF DMF FIRST MOLARS PER 100 PERSONS	NUMBERS OF FIRST MOLARS EXTRACTED AND REQUIRING EXTRACTION PER 100 PERSONS FIRST MOLAR MORTALITY	LOSS OF FIRST MOLARS PER 100 DMF FIRST MOLARS FIRST MOLAR LETHALITY
142	0	0	0
115	100	22	22±3.86
103	200	77	38±3.38
94	300	102	34±2.77
114	400	148	37±2.33

Therefore, we are more prone to believe that this uniformisation of first molar lethality figures is simply due to a longer exposure. Theoretically, tooth lethality is limited to 100% as only carious teeth are extracted and need extraction in the age brackets examined; in practice, this value cannot be attained. Thus, with longer exposure the tooth lethality curve becomes similar to what is called the saturation curve, this similarity manifesting itself in an equalization of the lethality figures accompanying at first higher, later also lower morbidity figures.

SUMMARY

The influence of tooth morbidity upon the loss rates of the first permanent molars was examined in the 10 to 11, 12 to 14, and 16 to 18 year old school population in three Hungarian urban communities. The number of teeth missing or in need of extraction per 100 decayed, missing or filled teeth (DMF) is called tooth *lethality*. Within the same population, tooth lethality increases with increasing prevalence of dental decay

in the younger age brackets. In the 16 to 18 year olds, however, there is less tendency for tooth loss to vary with the variations in the DMF.

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THICKENING OF THE SUBCUTANEOUS TISSUES IN PARALYZED LIMBS IN CHRONIC HEMIPLEGIA

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INTRODUCTION

WHILE measuring the thickness of the subcutaneous tissues of some male patients it was noticed that in some cases of chronic hemiplegia the measurements on the diseased limbs were greater than those on the normal side. In marked cases, the difference between the corresponding areas of the two sides could be readily appreciated by raising a skinfold on the patient's limb between one's thumb and index finger. In order to determine the extent and frequency of such changes, a statistical study of skinfold measurements of 34 male hemiplegic patients in the Infirmary of the Wayne County General Hospital, Michigan, was undertaken.

SUBJECTS AND METHODS

The patients had had hemiplegia for 10 months to 47 years, most of them from 3 to 10 years. Their age varied from 40 to 75 years; the average was 62.9 years. Eleven patients were bed patients. All were survivors of "strokes," with one exception; he had a brain injury 47 years before. During the investigation, tendon reflexes, wrist and ankle clonus, and the Babinski reflex were tested. A central motor disorder (spastic paralysis) was confirmed in each case.

The degree of paralysis of the muscle groups in the limbs was tested and recorded as strong, good, fair, poor, trace and zero (Daniels, Williams and Worthingham, 1956). The word "strong" is here substituted for "normal," because certain groups of muscles in hemiplegic patients "contract strongly against gravity and with full resistance," although movements of the limbs are not "normal." The following muscle groups were tested:

1. Extensors of the elbow.
2. Flexors of the elbow.

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3. Extensors of the wrist and the fingers.
4. Flexors of the wrist and the fingers.
5. Extensors of the knee.
6. Flexors of the knee.
7. Plantar flexors of the ankle and the toes.
8. Dorsiflexors of the ankle and the toes.

Skinfold calipers are commonly used to measure subcutaneous fat, especially in studies of growth, maturation and nutrition. The caliper used in this experiment is a British-made Harpenden skinfold caliper (as recommended by Edwards, *et al.*, 1955). It has a measuring surface of 6×9 mm on each jaw; the jaws are approximated by a spring that exerts a constant pressure of 10 gm per sq mm. The skin-fold thickness was measured to the nearest 0.1 mm. The measurement includes a double fold of skin and subcutaneous tissue.

Skin-fold measurements were taken on the normal and diseased sides of each patient over each of the groups of muscles mentioned as having been tested for muscle power. The 8 sites measured were as follows:

1. Back of the arm, half-way between the acromion and the olecranon, with forearm flexed at 90° (over the extensors of the elbow).
2. Front of the arm, same level as 1 (over the flexors of the elbow).
3. Back of the mid-forearm (over the extensors of the wrist and the fingers).
4. Front of the forearm, same level as 3 (over the flexors of the wrist and the fingers).
5. Front of the thigh, 7 inches above the upper border of the patella with the knee flexed at 90° (over the extensors of the knee).
6. Back of the thigh, same level as 5 (over the flexors of the knee).
7. Back of the leg, 3 inches below the skin crease opposite the knee (over the plantar flexors of the ankle and the toes).
8. Front of the leg, just lateral to the shin, same level as 7 (over the dorsiflexors of the ankle and the toes).

FINDINGS

The skinfolds were frequently increased in thickness, sometimes considerably, on the diseased limb of the chronic hemiplegic patients. The absolute and relative differences in skinfold thickness of the two sides

were calculated. The averages of these measurements are given in table 1. The relative thickening of the skinfold on the paralyzed limbs vary from 21.5 to 45.0%; it was most marked over the upper arm and least over the forearm and leg.

TABLE 1

Average thicknesses of skinfolds in different regions of the limbs of hemiplegic patients.

	AVERAGE THICKNESS OF SKINFOLD DISEASED SIDE MM	AVERAGE THICKNESS OF SKINFOLD NORMAL SIDE MM	AVERAGE INCREASE IN SKINFOLD ON THE DISEASED SIDE MM	AVERAGE RELATIVE INCREASE ON THE DISEASED SIDE [1] %
Arm, extensor surface	16.0	11.6	4.4	42.8
Arm, flexor surface	7.1	5.0	2.1	45.0
Forearm, extensor surface	6.4	4.7	1.7	35.4
Forearm, flexor surface	8.3	6.7	1.6	28.4
Thigh, extensor surface	19.5	15.2	4.3	37.2
Thigh, flexor surface	13.7	10.7	3.0	33.3
Leg, plantar flexor surface	11.7	9.9	1.8	21.5
Leg, dorsiflexor surface	9.4	7.3	2.1	33.6

[1] The average relative increase in skinfold is computed from the individual relative differences.

The degree of paralysis of different muscle groups is quite variable in any given patient. The upper and lower limbs of the affected side of the same patient may be quite different in the degree of muscular function retained. The extensor and the flexor groups of muscle of the same diseased limb tend to be more related to one another, but it is not uncommon to find that they also are paralyzed to quite different degrees. Owing to this fact, each skinfold and its corresponding muscular group are considered as one unit in the analysis of the degree of thickening of the skinfold in relation to the degree of muscular paresis. In the 34 patients, there are altogether 251 changes in skin-fold thicknesses matched with their corresponding muscular tests.[1] For these cases the degree of difference in skinfold thicknesses in the 6 different degrees of muscle function are shown in figure 1.

[1] A few measurements were difficult to take and the readings are not included. There were also three cases with lower limbs amputated; two cases had amputation before and one case after the onset of the cerebral vascular accident.

In some cases, the skinfold of the diseased side was actually slightly thinner. This reversed difference never exceeded 35%. Nevertheless,

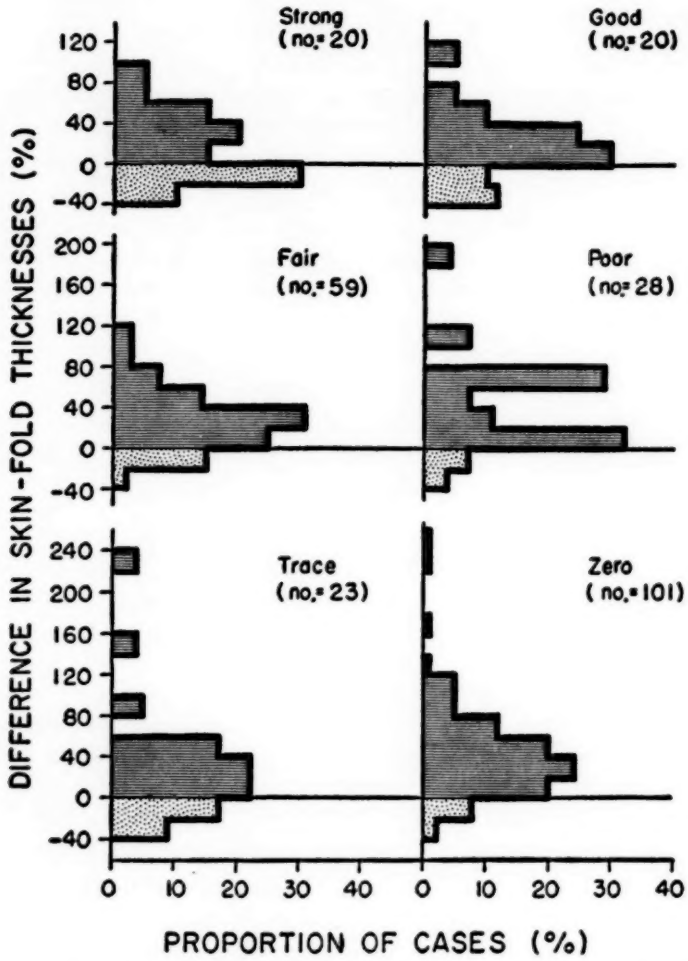

FIG. 1. DIFFERENCE IN SKINFOLD THICKNESS BETWEEN THE AFFECTED AND NON-AFFECTED SIDES ACCORDING TO THE DEGREE OF MUSCLE FUNCTION ON THE AFFECTED SIDE.

the existence of the biological variation which is reflected in this finding should make one wary of interpreting small differences as changes, at least in individual cases.

On the other hand, the most marked cases of thicknening of skinfolds occured in severely paralyzed limbs. In the 7 cases in which the measurement on the diseased side was over 120% of that on the normal side, the skinfolds all overlay muscles with poor, trace or zero muscle function. Furthermore, there was an average increase in the skinfold of the diseased side of 15.4, 18.9, 28.9, 44.1, 34.0 and 43.3% for strong, good, fair, poor, trace and zero degree of muscle function respectively. This reveals a tendency for more marked increase in skinfold thickening with more marked degree of muscle paralysis. The coefficient of correlation (r) of the degree of paralysis and relative increase in skin-fold is 0.21. This is not high, but the value is statistically significant ($P < 0.01$). To the extent that the evaluation of muscle function is not adequately quantitative and the categories not equal, this estimate of the linear correlation coefficient is conservative.

Duration of the paralysis is another factor that should be considered in respect to the degree of thickening of the skinfold. There are no acute cases to compare with the chronic ones, and the chronic cases can only be selectively compared with one another with exactly the same degree of paralysis. In the present series of chronic cases, there is no detectable correlation between the duration of the disease and degree of increase in skinfold thickness for any of the 6 degrees of muscular paralysis.

DISCUSSION

As was mentioned before, the skinfold thickness represents a combination of a fold of skin proper and the subcutaneous tissue. A thickened skinfold may be due to an increase in one or more elements in it. Skin, for instance, varies greatly in thickness. In a given area of the body, however, individual differences in thickness of the skin proper are small, especially when compared with the variablility of the subcutaneous fat (Richter, 1890, according to Brožek, 1950-51). The actual "feel" during palpation suggests subcutaneous fat increase rather than fibrosis, cellular proliferation or edema. The rate at which a constant tension caliper compresses a skinfold permits the user to some extent to distinguish fat from edema. Furthermore, the amount of tissue fluid could not greatly have affected the differences in the skinfold measurements because pressure from the caliper would have rapidly displaced extracellular fluid. Nevertheless, to substantiate the most plausible interpretation of the present findings, that most of the difference represents a change in subcutaneous fat, post-mortem investigation with gross and histological methods would be desirable.

Even without such confirmation the present study strongly suggests a relationship between the function of a group of muscles and the amount of overlying fat. It is a common notion that physical exercises of a part will avoid or reduce the local deposition of subcutaneous fat, and special devices for women, applying passive exercise to certain regions of the body, are supposed to be effective in local reduction of subcutaneous fat. There has been no direct evidence to support the principle, however, perhaps because of large individual differences such as those in hemiplegic patients.

Brožek (1952) explained that the thicker layer of subcutaneous fat in middle aged than in young adults may be due to decreased physical activities in the middle-aged group. He also measured one group of physically "active" and another group of "inactive" male individuals of the same age, and found that the subcutaneous as well as the total fat content of the body of the inactive individuals is higher than that of the active ones. Although nutritional surplus would be responsible for the high fat content in less active people; yet differential growth of body components also result in relative increase in fatty tissue, to the extent that muscular activities affect muscular development.

Buskirk, Andersen and Brožek (1956) found that in the unilateral active arm of tennis players there is an increase in the length of the long bones of the forearm and hand and stronger muscles on the active side than on the contralateral side, but unfortunately they failed to measure the subcutaneous fat in this study.

Unilateral increase in the subcutaneous fat in some cases of chronic hemiplegia is probably a result of lack of activity accompanying muscular atrophy due to disuse, but it is not specific to this kind of upper neuron paralysis. One victim of poliomyelitis was observed and he also showed considerable increase of subcutaneous fat over the atrophied muscles of the crippled limb.

SUMMARY

In 34 chronic hemiplegic patients the thicknesses of the subcutaneous tissue on the normal and the diseased limbs were measured with a skinfold caliper. Eight skinfold thickness dimensions averaged from 21.5 to 45.0% more on the diseased limbs than on the corresponding region of the normal limbs. The degree of thickening of the skinfold correlates significantly with the degree of muscle paralysis but there is considerable individual variability. The author discusses the suggestion that the unilateral increase in subcutaneous fat in some cases of chronic hemi-

plegia is probably a phenomenon resulting from decreased activity of the diseased limbs.

ACKNOWLEDGMENT

The author is most grateful to Doctors S. Jacobson and H. J. Wells of the Wayne County General Hospital, Michigan, for allowing her to study their patients. She is much indebted to Dr. G. W. Lasker of Wayne State University for his guidance and help with the statistical analysis and thanks are also extended to Dr. E. Gardner and Dr. F. A. Morin of the same university for reading and criticizing the manuscript.

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FURTHER STUDIES ON THE RELATION BETWEEN HEAD HAIR COLOR AND METALS IN HUMAN HAIR

A correction of the previous paper.

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IN 1956, I published in this Journal an article entitled "Relation between hair color and metals in human hair." This research was helped by two assistants. But at the end of 1956, it was discovered that our results were not reliable and we have been obliged to reexamine those of our previous experiments which were carried out by one of our former assistants. A portion of the necessary corrections was published in "Science."

Reexamination of the metal content of human hair was carried out by dry ashing and paperchromatography, but the results were not as clear as indicated in my previous paper. The wet ashing method and quantitative analyses of metals were also used on human hair. The outcome was also equivocal, *e. g.*, red, Fe 31 (ppm), Cu 49; blonde, Fe 17, Cu 11; brown, Fe 40, Cu 21; black (European), Fe 24, Cu 22; black (Japanese), Fe 59, Cu 29; black (Japanese), Ni 1.5; gray (Japanese), Ni 3.3.

These results indicate that my previous assumption of a correlation between hair color and metal content is incorrect.

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BOOK REVIEWS

Variation of External Body Form in the Individual. By W. W. Howells. 116 pp. Peabody Museum, Cambridge, Mass., 1957. No price indicated. Dittoed.

Professor Howells' monograph is an outstanding contribution to the study of human constitution. Its chief value lies in the highly ingenious methods of analysis rather than in the particular body types which come out of the study.

In the history of somatology, two somewhat divergent streams are apparent in writings both on race and on physique. One stream is largely descriptive and non-metric, and includes both racial typologies and somatotyping. In practice, somatotyping resembles racial classifications in being visual and impressionistic, whilst using standardized photographs instead of visual images. In order to make such descriptive systems more objective, measurements may then be introduced within the accepted framework provided by the types.

The second stream is largely atomistic and quantitative. Racial or constitutional variants are defined on the basis of individually measurable characteristics such as lengths of different parts of the skull and skeleton, and the various indices derived from these. In research on physique, the most elaborate atomistic methods have used devices, such as factor analysis, allowing the student to check "which characteristics go with which" instead of relying upon arbitrary impressions of the salient characteristics of groups of individuals.

Professor Howells' approach suffers neither from the arbitrary limitations of somatotyping nor from the atomism of trait analysis, and he is able to throw light on both. His method is to measure directly the degree of likeness between individuals—or rather, between a collection of measurements taken on the individual—since it is obviously impossible to include all of a person's characteristics. From the direct comparisons between persons (expressed in the form of correlations or in Howells' case, covariances) he obtains factors for persons in the way that trait factor analysis is used for grouping measurements. Howells then studies the measurements in respect to their ability to characterize the groups of individuals which he has already established. The individual comes first, the signs second, unlike the traditional factor method which studies the signs first and only proceeds to the individual (if at all) by the somewhat

laborious method of counting their weighted or unweighted scores in the different indicators. Howells finds that people are alike and then discovers the similarity to be in their bulkiness, tallness, shortness and so on.

Previously, Burt in England has used this double approach in studying physique, and examined the special conditions necessary for the results to be identical from which ever end they are applied: i. e., from persons or traits.

In any case, one must arbitrarily choose both the individuals and traits to be measured, and neither form of analysis can give results which are independent of these selections. Howells sampled 30 individuals to be as highly representative of white Army separatees as possible, and chose 50 measurements with the aim of characterizing body form with as little overlap as possible of lengths, diameters and circumferences. Weight, skinfold thicknesses and the dimensions of bones and other internal structures were not included.

All the men had previously been body-typed by the late Professor Hooton, and representativeness here refers to the distribution of these body types. This term does not apply so satisfactorily to the list of body measurements, which are somewhat biased by a preponderance of horizontal over vertical dimensions.

Covariances between each person and all the remainder were calculated by first expressing each person's measurements as deviations from the mean for that measurement, and then summing the deviation products obtained for any pair of individuals over the whole 50 measurements to give a single figure for the relation between each pair, so producing a 30 x 30 matrix of persons' relations. The table of covariances is then analysed into principal components, five of which are discussed. The first and most important component, which accounts for 54% of the total variance, corresponds to the major grouping of individuals. Second and subsequent factors correspond to the groupings left after extracting each preceding grouping in turn. These "person" factors are transformed to measurement factors by multiplying the measurements by the normalised person factors as weights. This method makes it possible to arrive at scores whose assessment depends on the dimensions rather than the individuals.

Factor 1 is strongly loaded with major body girths, while lengths and head measurements have very low weighting. When different combinations of measurements are compared for their efficiency in measuring this factor, the best 12 account for 84% of the variation of persons in

this factor. Further, the persons' weighted scores based on these 12 measurements correlates 0.99 with the full score obtained by using all 50 measurements—a very notable gain in economy.

Factor 2 defines centralization versus decentralization of mass (paunch and short limbs versus more strung out mass). It therefore does not correspond with the length-girth dichotomy usually found when factorising body measurements. I believe that this disagreement arises because there are too few lengths available to oppose the influence of the girths (despite their high negative loadings), and hence some girths must join their grouping. The six measurements which correlate most highly with the factor consist of trunk and limb lengths contrasted with waist, axillary arm and ankle girths, which is what Burt would have found.

As was true for factor 1, Howells arrived at efficient regression equations for factor 2 which used far smaller aggregates of measurements: in this case some six to twelve.

Factor 3 differentiates individuals with large chest to waist or hip relationship and the converse: i. e., taper from chest to hips with short limbs—especially in their distal segments versus slighter torso and longer limbs. This suggests a modified mesomorph versus leptosome distinction; fat and lean men are found in either group. Factor 3 differentiates chest size chiefly through muscle development: whereas factor 4 does so mainly through lateral skeletal growth.

Howells' emphasis is mainly on body form, and it is possible that had he used body measurements (such as skinfolds), indicative of body composition, or X-ray studies of bone, muscle, fat and viscera he might have slightly altered his interpretation of the factors. Whether this is so or not, Howells' mathematical analysis is quite unequivocal, and enough data are available in the Appendices to permit other forms of factor analysis to be attempted. Although Howells himself is not yet confident of the real nature of the factors emerging from his computations, he seems to have gone a very long way indeed towards making such a clarification possible.

The second part of the study concerns the relations between the measurements and the typing systems of Sheldon and Hooton, and really brings out the flexibility of Howells' method of analysis. The original constellation of men according to their measured similarities is fixed, and Howells' analysis has provided the principal (orthogonal) components, and it is now possible to examine the type ratings of these men on Sheldon's or Hooton's systems in relation to these axes.

The major axis resulting from both typing systems proved to be more closely related to the classic endomorphy than to the bulk factor of Howells: i. e., it partook also of some of Howells' second factor, but the ectomorphs do not fall at opposite ends of this axis. Whilst endomorphy and ectomorphy can be related to two axes, mesomorphy is not amenable to classification as an entity. Hence Sheldon's system is really two dimensional and the differences between Hooton's first component (fat) and Sheldon's endomorphy can be given. Sheldon's mass is concentrated at the equator (waist) level and rapidly falls outwards, whereas Hooton's corresponds to more general fullness. Hooton's first and third components are strongly opposed; Sheldon's ectomorphy and endomorphy opposed less strongly. Hooton's second component and also mesomorphy have to account for a considerable amount of variation, and the answer may be that both systems fail to take account of *general size* or bulk within which the opposing tendencies are present. Like Howells' earlier work, his present findings suggest that the whole system of somatotyping needs a shake up and a reformulation. Perhaps we on the other side of the Atlantic have been less ready to accept the whole basis of somatotypes than those closely connected with the details of the system.

I could not agree more with Professor Howells when he says "what is needed is some systematic approach which allows the body form to speak for itself, and to make its own statements as to the nature and relative importance of its several kinds of variation." Professor Howells' work has done just that most eloquently.

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Morphological Integration. By EVERETT C. OLSON and ROBERT L. MILLER. xv + 317 pp., 81 figs., 111 tables. The University of Chicago Press, Chicago 37, Illinois, 1958. $10.00.

Morphologically and functionally man is the best known of all vertebrates. In addition to the wealth of detailed descriptive anatomy that is available, the physiologists, functional anatomists and orthopedists have provided a tremendous mass of data on the functioning of the human body. The fossil record for the hominoids is still fragmentary,

but it is possible to consider in some detail the morphologic transition from primitive primate to man. In spite of this knowledge, there has been no attempt to analyze, from the standpoint of the integrated organism, the changes in the feeding and locomotor mechanisms that occurred during this transition.

Most functional studies deal with a single morphologic unit such as the femur or a single functional complex like the mandibular joint. The practical reasons for studying a complex organism by parts or systems are obvious, yet there is great danger in forgetting that evolution involves the reaction of the total organism with its environment. But the study of the total organism from a functional or even a purely morphologic point of view presents nearly insuperable difficulties. Olson and Miller have approached this problem in a highly original manner, even though the basic thesis is not a new one: the degree of interdependency (correlation) between morphologic components is directly related to the extent of the morphologic integration of these components.

Briefly, the method, which is entirely quantitative, involves taking a large number of predetermined measurements of a morphologic complex such as a skull, the axial skeleton, or the molar series of a mammal. Frequently hundreds and occasionally over a thousand measurements have been made in a single complex with population samples ranging from 10 to 40 (limited because of the labor involved in the computation or, in some cases, by available fossils). Correlation coefficients were computed for all measurements in pairs, followed by special statistical procedures designed for grouping the measures into "Rho-groups," as expressed by the correlations, and "F-groups" which are based on the Rho-groups and have biological, usually functional, significance. An index of morphological integration provides a numerical value for the extent of total integration (mean level of correlation for measured variants). Working with population samples of adult vertebrates (e. g., *Rana* and *Aotus*), with ontogenetic series (*Rana*), and with phyletic series of both vertebrates and invertebrates (*Pentremites* and *Hyopsodus*), the authors have investigated the inter-relationships of selected morphologic complexes (which they regard as more or less separately evolving units within the organism) in relation to growth, function, and, where appropriate, change through time. They have not attempted to explain some unexpected high correlations like the one between total length of pelvis and fibula length in the albino rat. Could this be the result of chance alone, or does it have some obscure developmental or functional

meaning? The relationship between function and changes in the evolving units has been considered for several examples. This approach should be particularly fruitful for studies on the evolution of man and other vertebrates with preserved hard parts.

The methodology developed by Olson and Miller for studying morphological integration is tedious and time-consuming, and it requires considerable sophistication in statistical manipulation. The statistics have been criticized (see Simpson, G. G., 1958, Science, vol. 128, p. 138) and some feel that the results are not worth the effort. There is little question, however, that the book will stimulate thought in new directions, and only time can tell what its impact may be on evolutionary biology. As the authors point out, this method of studying morphology makes it necessary "to cast thinking about morphology into the framework of the concept of covariance." This, in effect, is the basis for their interpretation of integration, an interpretation which, they believe, is not antagonistic to other modern approaches to morphological investigation.

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Physical and Behavioral Growth. Edited by PHILIP J. AMBUEL. 104 pp. Ross Laboratories, Columbus 18, Ohio, 1958.

This pamphlet reports the discussions of the 26th Ross Pediatric Research Conference held at the University of California School of Medicine in San Francisco, October 30-31, 1957. Although the 17 papers are quite brief, they introduce the reader to recent important advances along the broad spectrum of physical and behavioral growth and development.

One is impressed with the results steadily flowing from the longitudinal research programs, many of which are at least 30 years old. The power of the longitudinal method for the study of *process* is repeatedly demonstrated in the interrelationships which can be established among important variables measured in the same persons across time. In this context, individuals from a variety of professional disciplines are able to make contributions of considerable interest to one another, as shown in the brief discussions recorded at the end of each paper. Finally, impressive is the fact that much of the data can be translated into action

programs; the research, while not applied research, has obvious applications.

Hilda Knobloch points out the significance of environmental patterns for patterns of growth, particularly as the environmental variations relate differentially to disease conditions. Her finding of a much higher correlation between infant developmental schedules and later Stanford-Binet status for children from a disadvantaged group remains unexplained, as does Dorothy Eichorn's finding of a marked sex difference in body temperatures during the early adolescent years. Sontag opens up the fascinating problem of the impact of mothers' emotional disturbance on the behavorial development of the fetus, mediated possibly by physiological stress and hormone imbalance. Sears contributes a valuable paper bringing out the complexity of relationships in the attitudinal area. Sontag, Harris, Jean W. Macfarlane and Jones in separate papers show how complex this interrelationship can be in intellectual, physiological and physical growth, and in physical performance. What the infant and child does—how he behaves—constructs an environing world which in turn modifies further developmental changes.

In a concluding paper on the philosophy of growth, Frank raises the question as to whether we are struggling to comprehend the phenomena of growth in an inappropriate language. It is true that we are still utilizing concepts, terminology and statistical tools borrowed from physics, and especially mechanics. Yet growth seems to exhibit phenomena not readily reducible to mechanical concepts. The model for analysis of growth processes must of necessity be very complex. Yet it is possible that the basic patterns for the conceptual and statistical analysis of growth processes have been available for a long time; it is possible that our problem is chiefly one of handling the many factors involved. With electronic computers and new processes for handling masses of data and multiple variables at hand, the experimentalist need no longer be limited to analytical models which use single or at most three or four factors in multiple relationships. This model may look grossly oversimplified for what appears to an older generation of research workers as an intricately organized complexity. It may well be that a new generation of research workers, trained from the outset in the thinking that must accompany the operations made possible by high speed electronic devices, will be required to expand the concepts and to apply the resulting new ideas.

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Variables Related to Human Breast Cancer. By ELVING ANDERSON, HAROLD J. GOODMAN, and SHELDON C. REED. xiii + 172 pp. University of Minnesota Press, Minneapolis, 1958. $4.00.

The purpose of this study was to determine whether there is a significant excess of breast cancer in relatives of women selected because they have breast cancer (designated as propositae) and whether any familial tendency to cancer (if such exists) is site specific, or whether it is for cancer in general.

The authors selected the propositae from the Tumor Clinic of the University of Minnesota Hospital and chose the brothers and sisters of the propositae's husbands as controls. Factors such as age, marital status, fertility, residence (whether urban, rural non-farm or rural farm), nationality, etc., were considered. The information was secured by questionnaires sent out to relatives of the two groups, by personal interviews, and by use of death records and hospital reports.

The following criteria and procedures were used for the analysis: (1) proportional death rates (the percentage of deaths in individual age groups that are attributable to breast cancer); (2) mortality and morbidity rates in relatives; (3) a direct age-adjusted comparison between cancer and control groups; (4) a comparison of the morbid risks for cancer in the two groups; and (5) sequential analysis, applied to the data on sisters only.

With the exception of the sequential analysis method, all analyses showed a statistically significant excess of breast cancers among *sisters* of breast cancer propositae as compared with the controls. The *mothers* showed an excess of breast cancers only if the cases reported as breast cancer but not verified by official records were used.

To obtain information on *daughters* of breast cancer propositae a second group whose breast cancers were diagnosed about 25 years ago were chosen. This allowed a span of a generation during which their daughters might develop breast cancer. As a control for this group, the wives of the sons of the propositae were chosen. As the breast cancer rate is much higher in single than in married women, the single daughters were excluded from the group, and only married daughters were compared with sons' wives. They showed three times as much breast cancer as the controls.

This study, like all such researches, will prove of interest in its entirety mainly to the limited number of workers in the immediate field,

although some of the conclusions will be of general interest: Breast cancer probably includes a group of conditions heterogeneous in etiology, which may ultimately be separable into groups based upon pathological or biochemical differences, in which case genetic analyses will have to be made for each type.

The authors state that the genetic factors depend so much upon environmental factors for expression that it is difficult to demonstrate their effect. To the reviewer, the facts brought up in this volume do not support such a conclusion. If, however, the authors mean that it is difficult to demonstrate the *number* of genetic factors involved or the *mode of inheritance*, the reviewer is in complete agreement.

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The Aging of Populations and its Economic and Social Implications. United Nations (Population Studies No. 26), vii + 168 pp. New York, 1956. $1.75.

In order to avoid any overlapping with functions of the specialized agencies of the United Nations, this report specifically disclaims any intention to deal with employment of older persons or the medical aspects of aging. Thus, it is strictly limited to the field implied by its title.

It is composed of 4 chapters, dealing with "trends and differentials in the aging of populations," "causes of the aging of populations," "economic and social implications," and "future trends," together with an introductory chapter and an appendix in which detailed statistical data are given. Different kinds of aging of populations are distinguished by referring to the "age pyramid" which is conventionally used in census reports and demographic texts to represent the age distribution of a population. Thus, an increase in the proportion of aged persons is described as aging "at the apex" and a decrease in the proportion of young persons is described as "aging at the base."

Contrary to popular belief, the reduction of mortality as it has occurred historically has had little effect on the age structure of populations. It has led to an increase in the proportion of young persons and

has left the apex of the pyramid practically unchanged. On the other hand, the reduction of fertility has led to considerable aging both from the base and at the apex. For the time being, thanks to the advances of medical science and to economic and social development, the underdeveloped countries are experiencing a reduction of mortality without any decline in fertility. There is thus a rejuvenation from the base, the apex of the pyramid remaining unchanged. It is reasonable to suppose, however, that mortality will not continue to decline indefinitely without leading to a decline in fertility which will give rise to heavy aging from the base and at the apex. It can, therefore, be forecast that in the underdeveloped countries a phase of rejuvenation will be followed by a phase of heavy aging. A process of this kind is now occurring in Japan.

Chapter 4 is devoted to the study of future developments in the countries which are in the vanguard of demographic aging, that is, those with low fertility. In most of these countries, the decline in fertility, which began over a century ago, seems to have halted. It has been shown that the decline in fertility was responsible for demographic aging and it might be expected that demographic aging would now slow down. However calculations show that the full effects of the decline in fertility in previous decades have not yet been felt in the age structure of the populations and, although fertility has been stabilized, the populations will continue to age for some time. Moreover, although the decline in mortality in the form in which it has occurred has caused little change at the apex of the pyramid, there is reason to believe that this will not be the case in the future. If mortality continues to decline in countries where it is already low—and there is every reason to hope this will be so—it will decline particularly in the upper age groups. The rates in the younger age groups are now so low that it would be unreasonable to hope for appreciable gains at those ages. The low-mortality countries are also those where fertility is low. It follows that in those countries a future decline in mortality will normally result in aging of the population.

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The Fertility of American Women. By WILSON H. GRABILL, CLYDE V. KISER and PASCAL K. WHELPTON. xvi + 448 pp. Wiley, New York, 1958. $9.50.

Social and Psychological Factors Affecting Fertility. Vol. 5. Edited by P. K. WHELPTON and CLYDE V. KISER. vi + 285 pp. Milbank Memorial Fund, New York, 1958. $1.00.

The first of these books is devoted to an examination of the nature of the factors affecting trends and variations in human fertility in America from Colonial times to the present. Most readers will probably be surprised to learn that from the Colonial and early Federal periods American fertility has been among the world's highest.

The immense amount of data is reduced to manageably useful proportions by analyzing them by century, year, residence, ethnic group, socio-economic status, education, marriage and child spacing, and so on. The authors seem to have missed nothing, and the numerous tables and figures are stuffed full with illuminating facts. If there is a criticism it is that the explanation of terms is not quite comprehensive enough. There is a summary, a fine bibliography, and an index.

I do not think it is too much to say that this is probably the most satisfactory monograph on American fertility to be published in our time.

The second volume represents the concluding reports and summary of the chief findings from the Indianapolis study. This pioneering study to learn something of the social and psychological components of human fertility was initiated in 1938. Twenty-four articles reporting the results and analyses of hypotheses have already been published. In this fifth and final volume of reports Drs. Edgar F. Borgatta and Charles F. Westoff open with a discussion of scaling techniques and factor analysis as applied to the materials for all couples and for planned families. Drs. Ruth Riemer and P. K. Whelpton report on the attitudes of women toward personal freedom in relation to fertility—a report which shows the inherent difficulties in such studies, but nonetheless valuable for all that. Potter and Kantner report on hypotheses relating to childhood situations and conformity to group patterns, and test some reformulated hypotheses on the influence of siblings and friends on fertility. Pratt and Whelpton discuss the hypothesis concerning interest in and liking for children, and extra-familial participation of wives in relation to

interest in children, fertility planning and size of planned family. Solomon, Clare and Westoff present data on two hypothecated reasons for the second child, and the data on the more general hypothesis that the interest of children in, and their desire for, brothers and sisters affect the size of the family. Musham and Kiser discuss the data on children desired by wife and husband at the time of marriage in relation to fertility planning and actual number of children 12-15 years after marriage. In the final article the editors summarize the outstanding findings of the Indianapolis Study and discuss some of the methodological lessons for future investigators.

If the Study opened up more questions then it answered, it nevertheless managed to throw a considerable amount of light upon the nature of the problems and the processes involved. It was a study well worth undertaking, and has been brought to a conclusion in a manner which deserves our thanks and congratulations.

ASHLEY MONTAGU

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Of Stars and Men. By HARLOW SHAPLEY. vii + 157 pp. Beacon Press, Boston, 1958. $3.50.

"I don't pretend to understand the Universe—its a great deal bigger than I am . . . People ought to be modester." Thus spake Carlyle. Dr. Harlow Shapley has a section in the last chapter of this encouraging book entitled "Humility Comes Naturally" which, however, gives little or no support to Carlyle's dictum. But then, Carlyle was a pessimist, while Shapley is an unreconstructed optimist. In this quite fascinating volume Shapley shows us that in the moment of Time in which the human species has existed it has already come to know an astonishing number of things about the un-Carlylean universe, and that there is every promise—providing we last long enough—that we shall know a great deal more. We may not ever know everything about everything, but there is some chance that we may eventually come to know everything about some things. But that is not the burden of Shapley's message. Shapley wants us to achieve a proper perspective on ourselves. Copernicus turned us from geocentricity to heliocentricity, in our own time we have advanced toward a certain degree of anthropocentricity. Shapley, I suspect, wants to ward off the advance to egocentricity of which so

many of us, whose brains so to speak have gone to our heads, show dangerous evidences. Toward this end Shapley shows us how incidental our little world is in the cosmos, that in all the millions and millions of galaxies we are on the very periphery of the outskirts of a quite ordinary galactic system, and that it is highly probable that not only are we not the only form of intelligent life in the universe, but that the universe simply teems (or is it "bristles"?) with vast numbers of intelligences occupying vast numbers of different planets. A conservative estimate would put the number of such biotic planets at not less than one hundred million. There is not the least reason to doubt that some of these intelligences may be a bit brighter than *Homo sap.* They may be endowed with all sorts of senses that we do not even dream of. In short, the probabilities are that in the metagalactic universe we are not alone. I somehow find this a comforting thought. It is something to look forward to: getting to know our neighbors in the metagalaxy.

The probabilities of any extra-terrestrial forces putting an end to this planet are quite as low as those for the terrestrial ones, with the one exception of man himself—man's worst enemy. For this reason, though it may not be generally known, Shapley has always been one of the most active supporters of the social sciences, for as he realizes, it is more important just now to get to know something about the inner constitution of man than it is about the inner constitution of the stars.

Plutarch tells us that when Alexander heard from Anaxarchus that there was an infinite number of worlds, he wept. When friends inquired the cause of his tears, he made answer: "Do you not think it a matter of lamentation that when there is such a vast multitude of them, we have not yet conquered one?" We have yet to conquer ourselves. Can we do it? Of course we can, but the question is, will we? Books such as Shapley's make us feel that it is very much worth trying.

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Princeton, New Jersey

A Textbook of Psychology. By D. O. HEBB. 276 pp. W. B. Saunders, Philadelphia, 1958. $4.50.

The Behavior of Man, an Introduction to Psychology. K. U. SMITH, and W. M. SMITH. 516 pp. Henry Holt, New York, 1958. $6.95.

It is not ordinarily the policy of HUMAN BIOLOGY to review textbooks. But these are not ordinary textbooks. Your book review editor saw a unique opportunity in requesting a comparison of these two contributions to the growing scientific study of the biology of behavior. Hebb's compact book is an innovation or reversal of trend in the introductory psychology field, and to the casual browser it looks like a study in contrasts when placed beside the Smith and Smith volume. The Smith brothers have achieved a distinctive and distinguished innovation in visual education.

The central point of view of these two books is as nearly identical as independent authors are likely to achieve at this youthful stage of psychological science. They write with a broad scope, yet carefully select within each topical area those experimental and observational data which support theoretical generalizations. The emphasis is on what Hebb calls the method of *behavioristics.* Both books are thoroughly up to date. They do not slight any of the major methods of investigation. Description, quantitative measurement, experimental methods, and correlational techniques are all represented both in sections dealing specifically with the questions of methodology and in a rich coverage of empirical findings. It is difficult for any worker in psychology, and much more so for persons in related biological and social sciences to keep abreast of current research in each of the several major areas of study. Either of these texts can bring the reader an overview of current knowledge which should substantially reduce his feeling of being "out of touch" with the newer developments.

Both books draw freely from the experimental literature on animal behavior, and from physiological psychology regardless of the organism used for the investigation. The development of human behavior is a topic explicitly recognized in chapter headings of each book. Hebb's title for this coverage is: "Heredity, maturation, early learning." He reaches this topic in his sixth chapter, following two chapters on definitions, methods, and interrelations between concepts, and three chapters on neurophysiological mechanisms in behavior. Smith and Smith follow a similar organization, placing their explicit discussion of bodily pro-

cesses in chapter 4, to precede their treatment of the development of behavior. The close relationships between behavior and its organismic foundations are manifest through most of the remaining 13 chapters of Hebb and 17 chapters of the Smith book.

These two books are fundamentally suited to the same purpose: to tell the scientific story of behavior to those who have only the general liberally educated layman's acquaintance with it. Smith and Smith have found a little more space than Hebb to devote to the topic of frustration and to the practical problems of adjustment. They have also included more descriptions of experiments, and their index includes a somewhat larger number of names of investigators. But even with this they relegate suggested readings, study questions, and projects to a Workbook designed to supplement the text. Original sources for materials discussed and illustrated in the text are listed by chapter. In this respect both books are fully self-contained and most satisfactorily documented.

There are a few major differences of emphasis between the two texts. Hebb has more to say about recent work on the "arousal system." He is brief in his explicit treatment of personality, covering this topic in 11 pages, whereas Smith and Smith gave it four chapters (Part III of their book). They also devote separate chapters to "thinking" and "verbal behavior." Hebb gives a brief discussion of communication and language as a small section within his chapter on "problems relating to thought." Those readers who know Hebb's research and his theoretical writings will find brief expositions of concepts which he developed in detail elsewhere. The "cell assembly" construct and neurophysiological assumptions concerning the diffuse conduction that underlies higher brain processes are explained in sufficient detail early in the book to permit reference to these in later chapters. A continuity is achieved in this way between sensory processes, learning, perception, and thought. The basic assumptions deal with how brain processes are affected by use. Smith and Smith deny that these assumptions can be put to direct test and Hebb is in full agreement. The choice that Smith and Smith make is to stay closer to the classical data of psychophysics, while Hebb bluntly states his position: "Current physiological knowledge is of course incomplete, and some flexibility is needed when we apply it to psychological problems" (p. 103). In his final chapter on scientific method he further defends neurologizing, as he is also quite willing to call it, as a sophisticated statement of ignorance, whose chief function is to permit tentative statements of relations between facts which must ultimately be inter-

related, so that this ignorance can be reduced. The facts calling for this relating are, of course, the fact of behavior and the other facts of biology. With characteristic candor Hebb asserts that man shows less need for prefrontal cortex than does monkey. This is obviously a challenge to those engaged in the development of tests for higher brain functions.

A final word is in order about the innovation of Smith and Smith. Their use of visual art achieves a good deal of what they wanted. Illustrative drawings and photographs give meanings to obscure bodily processes, abstract generalizations, and quantitative relations. To the sophisticated student they may often be superfluous, but to the less experienced they will enliven greatly a subject that is complex and fraught with difficulties of vocabulary.

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BOOK NOTICES

USES OF EPIDEMIOLOGY. By J. N. Morris. viii + 135 pp. William & Wilkins Co., Baltimore, 1957. $4.00. As the author indicates in the Preface, a more accurate title of this book would be "Some Uses of Epidemiology in the Study of Non-Communicable Diseases." This makes the volume a good deal more relevant to students of human biology.

FOUNDATIONS OF EMBRYOLOGY By Bradley M. Patten. xvi + 578 pp. McGraw-Hill Book Co., New York, 1958. $10.50. Written with the objective of furnishing a suitable text for a first course in embryology at collegiate level, the volume stresses the similarity of the major developmental processes in the vertebrates. Abounds in informative illustrations.

CLINICAL EPIDEMIOLOGY. By John R. Paul. xii + 291 pp. University of Chicago Press, Chicago, 1958. $5.00. An introduction to epidemiology, defined here as a science concerned with the *circumstances* under which people get sick or remain sick. This includes not only the microbiological and toxicological circumstances, but also genetic and somatic factors (including obesity and body build), and may require the scrutiny of social, religious and political influences.

PERCEPTANALYSIS. By Zygmunt A. Piotrowski. xvii + 505 pp. Macmillan, New York, 1957. $6.75. As the subtitle indicates, this volume presents "a fundamentally reworked, expanded, and systematized Rorschach method," a projective technique used widely for evaluation of personality in the clinical context and extended to field studies.

PRINCIPAL NERVOUS PATHWAYS. By Andrew Theodore Rasmussen. ix + 73 pp. Macmillan, New York, 1952. $5.50. A reprinting of the 4th edition of the well known neurological charts and schemata.

PSYCHOENDOCRINOLOGY. Edited by Max Reiss. vii + 208 pp. Grune & Stratton, New York, 1958. $7.00. Proceedings of a symposium arranged with the aim to appraise recent progress made in the elucidation of endocrine function in psychopathology, both in regard to therapy and to pathogenetic analysis of mental disease.

PARAPSYCHOLOGY: FRONTIER SCIENCE OF THE MIND. By J. B. Rhine and J. G. Pratt. ix + 220 pp. C. C. Thomas, Springfield, Ill., 1957. $4.75. A survey of the field, the methods and the facts of extrasensory perception and psychokinesis research, presented as a one-volume summary of present knowledge about parapsychology.

COMMUNICATION, ORGANIZATION, AND SCIENCE. By Jerome Rothstein. xcvi + 110 pp. Falcon's Wing Press, Indian Hills, Colo., 1958. $3.50. A physicist's excursion into philosophy of science, with information-organization theory as the core and sallies made into several problem areas (scientific theory as a system for prediction, physical models of behavior of organisms, and the possibility of constructing a universal language).

JUVENILE DELINQUENCY. Edited by Joseph S. Roucek. 370 pp. Philosophical Library, New York, 1958. $10.00. A collection of surveys, written by specialists and concerned with the various facets of a complex and signifi-

cant social problem. The 4 sections analyze the dimensions of the problem and the legal aspects, causes (including considerations of the biological basis of juvenile delinquency, pp. 59-78), solutions and international trends.

INSTINCTIVE BEHAVIOR: THE DEVELOPMENT OF A MODERN CONCEPT. Transl. and edited by Claire H. Schiller. xix + 328 pp. International Universities Press, New York, 1957. $7.50. Selection of papers documenting the approach and accomplishment of leading European students of "ethology," the comparative study of animal behavior, with special emphasis on environmental influences activating and modifying innate action patterns.

ESP AND PERSONALITY PATTERNS By Gertrude Raffel Schmeidler and R. A. McConnell. xiii + 136 pp. Yale University Press, New Haven, 1958. $4.00. A serious, methodologically sound contribution to the study of the process referred to as "extrasensory perception." Focussed on personality characteristics in relation to performance in a card-guessing task.

TESTING OF NEGRO INTELLIGENCE. By Audrey M. Shuey. xv + 351 pp. J. P. Bell Co., Inc., Lynchburg, Virginia, 1958. $4.00. A detailed summary of about 240 studies on Negro-White differences in mental-test performance, carried out in the U. S. and covering a span of over 40 years.

PRINCIPLES OF GENETICS. By Edmund W. Sinnott, L. C. Dunn, Theodosius Dobzhansky. xiv + 459 pp. McGraw-Hill Book Co., New York, 1958. $6.75. Revised edition (1st ed., 1925; 4th ed., 1950) of an outstanding perennial senior-graduate textbook. A chapter on Statistical Inference in Genetics was contributed by H. Levene. Human heredity is given due consideration.

ESSENTIALS OF HUMAN ANATOMY. By Russell T. Woodburne. viii + 620 pp. Oxford University Press, New York, 1957. $12.50. Stimulated by the need for a re-examination of the teaching methods in anatomy, resulting from the steadily increasing knowledge in the basic medical and clinical sciences, the author provides a textbook which presents—concisely but without faulty oversimplification—the basic features of the systems of the body, followed by a detailed regional presentation of body structure.

DYNAMICS OF BEHAVIOR. By Robert S. Woodworth. x + 403 pp. Henry Holt and Co., New York, 1958. $5.00. Appearing 40 years after the author's *Dynamic Psychology* and supplementing his classical portrait of *Experimental Psychology* (1938, 1954), the volume covers motivation, learning, perception, and problem solving, with emphasis on their interaction.

MEASUREMENT OF LEVELS OF HEALTH: REPORT OF A STUDY GROUP. World Health Organization Technical Report Series No. 137. 29 pp. Columbia University Press, New York, 1957. $.30. Tackling an important but difficult task, the study group classified the *health indicators* as those provided by vital statistics (crude death rate, infant mortality, deaths from communicable diseases, life expectation), related to relevant aspects of physical environment, and concerned with health services (such as availability and use of hospitals). Nutrition and morbidity studies were among the additional indicators recommended for study.

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